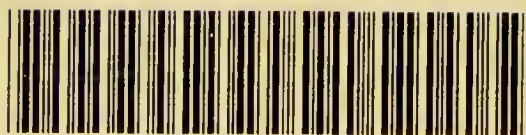


DISEASES  
OF THE  
EYE.

DR. CECIL SHAW,

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DISEASES OF THE EYE.

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# DISEASES OF THE EYE:

A PRACTICAL HANDBOOK FOR THE USE OF  
GENERAL PRACTITIONERS & STUDENTS,

BY

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Medical Society.*

WITH ILLUSTRATIONS AND A TEST CARD  
FOR COLOUR BLINDNESS.

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TO  
M. LE DR. EDOUARD LANDOLT,  
PARIS,

THIS BOOK IS DEDICATED  
IN ADMIRATION OF HIS CONTRIBUTIONS TO THE SCIENCE  
OF OPHTHALMOLOGY, AND IN GRATITUDE FOR  
MANY ACTS OF PERSONAL KINDNESS.



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## PREFACE.

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The aim of this little book is to give to the general practitioner and student a brief account of the principal affections of the eye and their treatment in as concise and practical a manner as possible. To this end accounts of the anatomy and physiology of the eye and of its physical optics have been omitted, as these subjects are best studied in the text books on Anatomy, Physiology, and Physics. Full directions for the various operations on the eye have also been omitted, for only a very small proportion of medical men undertake these, and for such there is an ample choice of large text books on Ophthalmology. Lastly, directions for the use of the ophthalmoscope have been omitted, as after several years' experience with students I am most strongly of opinion that its use can only be learnt from clinical teaching, and that all printed instructions are little better than wasted labour.

These omissions have made it possible to include fairly full accounts of all the commoner affections such as are likely to be met with in general practice, and also a special chapter on affections of the eye in connection with general diseases, which it is hoped will prove useful. This chapter was suggested to my mind by memories of the excellent course of lectures on this subject delivered by Dr. Dimmer, of Vienna.

My friend, Dr. Calwell, Senior Physician to the Ulster Hospital for Children and Women, has given me valuable suggestions and help in correcting the proof sheets, and to him I offer my warm thanks.

14 COLLEGE SQUARE EAST,  
BELFAST, *January*, 1895.



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## CHAPTER I.

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### THE EYELIDS AND LACRYMAL APPARATUS.

#### THE LIDS.

Ophthalmia Tarsi (Tinea Tarsi, Blepharitis Marginalis)—A chronic disease affecting the margins of the lids, characterised by an eczematous eruption and the formation of yellow crusts, matting together the cilia into bunches.

When the crusts are carefully removed the underlying surface is found to be red and inflamed (Blepharitis squamosa) or, in severe cases, ulcerated (Blepharitis ulcerosa). In the latter cases small abscesses form at the roots of the cilia, which are thereby destroyed or misplaced.

Causes—Generally constitutional. The great majority of cases are found in children enfeebled by tuberculosis or some of the exanthemata; both eyes being affected. Where one eye only is affected, a local cause may be found, commonly in a chronic inflammation of the conjunctiva or cornea.

Consequences—If neglected, it is likely to lead to complete loss of the cilia, and great disfigurement, or to their misplacement (distichiasis or trichiasis), which leads to irritation of the cornea by their rubbing against it, and subsequent inflammation and injury to vision.

Treatment—Warn the patient or his parents that treatment must be prolonged; if stopped soon, the case relapses. All crusts must be cleared off, night and morning, with a weak alkaline lotion (a teaspoonful of baking soda to a pint of hot water—may be made at home) and a piece of

soft rag or cotton wool, then the affected surfaces lightly smeard with a weak mercurial ointment (Hydrarg. Ox. Flav., gr. v; Vaseline, ži).

**Hordeolum** (Stye)—An acute suppuration of a sebaceous gland at the margin of the lid, characterised by a small swelling on the edge of the lid with severe pain, and a great tendency to recur in crops.

Causes—Anæmia, scrofula, and local inflammations predispose to the formation of styes. Generally found in young people, and sometimes from sexual disturbance. It is an indication of a poor state of health.

Treatment—Hasten the formation of pus by the use of warm damp compresses, and as soon as a yellow point is seen on the margin of the lid, open it with a needle or fine knife. To prevent recurrence, rub a very weak white precipitate ointment on the edges of the lids daily for a length of time, and use tonics.

**Chalazion** (Tarsal or Meibomian Cyst)—A cystic enlargement of a meibomian gland, causing a slowly growing tumour in the lid. There is no pain, and attention is only called to it by the swelling.

Cause—The cause is probably always a slight local inflammation, which may pass off before the tumour is noticed. Seldom seen in children ; generally in adults.

Treatment—When the tumour attains to the size of a pea, or even less, the lid should be everted, the conjunctival surface brushed with a cocaine solution and incised where the tumour is most prominent, and the contents, generally creamy or grumous in consistence, squeezed or scraped out—some roughness being necessary, so that the cyst walls may be broken down.

**Herpes** may occur on the lids, sometimes affecting the cornea also. It is recognised by the severe pain preceding it, the character of the eruption, and the fact that

it does not cross the middle line of the face. Arsenic and quinine may be given internally, and cocaine drops locally to allay the pain. If ulceration of the cornea occurs, it must be treated as such—*vide infra*.

**Lupus, Chancres, Epithelioma, and Sarcoma** are sometimes found on the lids, but do not present any peculiar characters in that situation.

**Ptosis**—drooping of the upper lid—may be congenital or acquired, and complete or partial, and may affect one or both eyes.

**Causes**—Congenital ptosis is bilateral, and is due to weakness or absence of the levator palpebrae superioris; acquired ptosis, which generally affects one eye only, to paralysis of that muscle. The latter is therefore often only one symptom in a group, as in Ophthalmoplegia Externa, where all the external muscles of the eye are paralysed. 370 H.

**Treatment**—In recent cases the usual constitutional treatment may be tried, and failing this, an operation to shorten the lid may be of benefit. The treatment of the congenital form is highly unsatisfactory.

**Distichiasis and Trichiasis**—When the cilia are arranged in a double row, it is called distichiasis; when irregularly, trichiasis. Either condition may be congenital or the result of diseases of the lid. The misplaced cilia, if they rub against the cornea, soon cause irritation and inflammation. Temporary relief may be obtained by plucking them out with forceps (epilation), while a radical cure is effected by destroying the roots of the cilia by electrolysis, or by a plastic operation on the lid altering the direction of its margin.

**Entropion**—A turning in of the margin of the lid, by which the cilia are brought into contact with the cornea. Entropion may be *muscular* or *organic*; the former

affects the lower lid only, the latter may affect either or both.

*Causes*—Muscular or spastic entropion is due to a contraction of the palpebral part of the orbicularis. This may occur after prolonged closure of the eye, as in severe cases of phlyctenular ophthalmia in children, and in eyes which have been bandaged for some weeks ; specially in old people, where the skin is loose. Organic entropion may arise from any cause altering the normal structure of the lids ; generally cicatrices. Burns of the conjunctiva with lime cause exceedingly troublesome entropion.

*Treatment*—Organic entropion can only be cured by plastic operations. Muscular entropion can, in most cases, be cured by adopting means to keep the lid turned out for a time, such as strips of plaster drawing it down, or several coats of flexible collodion painted on the lid after carefully drying the skin. In children frequent bathing of the face with cold water tends to lessen the contraction of the muscle. In old people relief may be afforded by the wearing of rather heavy spectacles, the lower edges of which rest on the lids and draw them down.

**Ectropion**—A turning out of the margin of the lid, varying from slight displacement outward to complete eversion of the lid. Like entropion, it may be muscular or organic. It is very unsightly, if at all extensive, and causes trouble by displacing the canaliculi, so that the tears flow down the cheeks instead of passing off by their normal passage.

*Causes*—Muscular ectropion may be—(a) Spastic, due to irregular contraction of the orbicularis, generally in connection with inflammatory swelling of the conjunctiva ; (b) Senile, from atrophy of the orbicularis ; (c) Paralytic, from paralysis of the same muscle. Organic ectropion is cicatricial, from wounds, burns, lupus, &c.

*Treatment*—Spastic and senile ectropion are treated by bandaging the eye, and if the conjunctiva is inflamed or swollen, brushing it with some astringent. The paralytic form is treated by electricity, and the organic by plastic operations.

---

## THE LACRYMAL APPARATUS.

The lacrymal gland is rarely affected by disease, being only occasionally the seat of malignant tumours. The apparatus for discharging the tears is often affected.

**Epiphora**—escape of the tears on the cheek—may be due to displacement of the punctum by entropion or ectropion, or to stenosis of the punctum or canaliculus. Stenosis of the punctum may arise from inflammation or injury to the surrounding tissue. A very fine probe should be tried, and if this fails to pass, the canaliculus must be slit up with one of the knives made for the purpose, such as Weber's. Epiphora also follows the undermentioned affections of the lacrymal apparatus.

**Stricture of the Nasal Duct** may be caused by cicatrices or swelling of the mucous membrane, or by disease of the bone. The last is seldom curable. The canaliculus is slit, and probes passed daily, beginning with the smaller sizes. Some few cases of tough cicatricial stricture have to be divided with a knife, such as Schmidt-Rimpler's, which may be used both for this purpose, and opening the canaliculus. After division, a large probe is passed to prevent contraction, at first daily, and then at longer intervals.

**Chronic Dacryocystitis** (Mucocoele)—A chronic inflammation of the lacrymal sac, characterised by obstruction to the passage of tears, and a mucous or muco-purulent

discharge, which escapes from the punctum on pressure being made over the sac.

*Causes*—When any cause can be found, it is generally in an inflammation extending to the sac from the mucous membrane of the eye or nose.

*Treatment*—Inflammation of the conjunctiva or nasal mucous membrane must be treated if present. A very fine pointed syringe should be inserted in the punctum (which must be dilated with a probe if necessary), and some fluid gently forced in. If it passes through to the nose, a cure may often be effected by regular syringing with some antiseptic and astringent fluid. If, however, there is an obstruction to its passage, the canaliculus must be slit up and a probe passed, after which it can be syringed daily, with occasional passing of the probe—as for Stricture, *vide supra*.

**Acute Dacryocystitis**—An acute purulent inflammation of the lacrymal sac and the tissues round it. The skin over the sac, under the inner angle of the eye, becomes red, swollen, tense, and very painful; and the whole surrounding parts, including the lids, are swollen and œdematous. The pain is often so severe as to prevent sleep, and there may be considerable fever. If left to itself the abscess soon points and opens on the cheek, and may remain patent after all acute symptoms have passed off (lacrymal fistula).

*Causes*—Cold alone may be accountable in some cases, but generally acute dacryocystitis occurs as a sudden aggravation of the chronic form.

*Treatment*—If the case comes under treatment at a very early stage, it may possibly be checked by slitting the canaliculus and syringing out the sac freely with antiseptic fluids. When once the inflammation is well under weigh this is hopeless, and we then encourage suppuration

by warm moist compresses, and open the abscess as soon as fluctuation can be recognised. The artificial fistula is kept open till all signs of inflammation are gone. If a stricture should be present it must be treated, and when the whole course of the normal tear passage is clear, the artificial passage generally heals of itself—or may be induced to do so by freshening its sides.

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## CHAPTER II.

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### THE CONJUNCTIVA.

The classification of inflammatory diseases of the conjunctiva presents considerable difficulties, as each form we may honour with a separate name, and consider distinct, will, nevertheless, be found to merge into other forms. The difficulty is further increased by the fact that the secretion from a particular form will often, if carried to another eye, produce not that form but a milder or more acute. It is, therefore, necessary to warn the student that, though the following classification will be found useful for clinical purposes, he must not take it as indicating the existence of a series of separate and distinct affections, like the specific fevers, for instance, but must be prepared to meet cases which may with equal justice be assigned to either of two classes. The classification we shall adopt is as follows :—I. Simple Conjunctivitis, with its sub-divisions—(a) Chronic, (b) Acute, (c) Follicular.

II. Purulent Conjunctivitis. III. Croupous Conjunctivitis. IV. Diphtheritic Conjunctivitis. V. Granular Conjunctivitis. Conjunctivitis lymphatica, or Phlyctenular Ophthalmia, which is as much a corneal as conjunctival affection, will be reserved for a separate chapter.

**I. Simple or Catarrhal Conjunctivitis—(a) Chronic.** A chronic inflammation of the conjunctiva, affecting chiefly the lids, characterised by slight redness and a very small amount of secretion, and by marked subjective symptoms. The redness may be so slight as to be almost unnoticed, and the conjunctiva remains quite smooth. The secretion is so small in amount that it only shows itself by glueing the lids together in the mornings, and it is on account of the subjective symptoms that the patient seeks relief. These symptoms are very various, but chief among them are pain, heaviness, and a feeling of heat in the lids, specially in the evenings; a feeling as of small grits or grains of sand under the lids; slight photophobia and excessive watering of the eyes; and occasional dimness of vision, from threads of sticky secretion on the cornea. The severity of these symptoms bears absolutely no relation to the strength of the objective signs of inflammation which are present. Chronic Conjunctivitis tends to run on for years, getting better and worse occasionally.

**Causes**—In some cases it is the result of an attack of Acute Conjunctivitis, which has slowly subsided but has never disappeared. More usually its onset is gradual, and in middle life and old age it is exceedingly common—arising specially from bad air. Alcoholic excess and digestive troubles, and probably a gouty or rheumatic diathesis, predispose to it; as also does straining of the sight. The most troublesome case that has ever come under my observation was typical as regards cause—a Customs officer, middle aged, working all day in underground

bonded stores, where the air was full of dust and alcoholic vapour, and book work had to be done by dim gas light.

*Treatment*—Few things are more unsatisfactory to treat than Chronic Conjunctivitis often is, for the simple reason that we cannot combat the causes. In the case just mentioned, for instance, it would be absurd to tell the patient that he must change his employment; and local treatment is not likely to cure while the existing cause remains active. But even where a cure can not be effected, relief is given by mild astringent lotions, such as Acid. Boric., gr. xv—ʒi, with or without Zinc. Sulphat., gr. ii, added. In the more severe cases, or during exacerbation in the course of milder forms, the lids may be everted and brushed daily with Argent. Nit., gr. x—ʒi. To prevent the lids sticking together in the mornings, any simple ointment may be rubbed along their edges at night. The sight should always be examined, and glasses ordered if necessary.

(b) **Acute Catarrhal Conjunctivitis**—An acute inflammation of the conjunctiva, characterised by marked hyperæmia and swelling of that membrane, with sticky secretion. The conjunctiva is affected in its whole extent in severe cases, but often on the lids only in milder. Photophobia is not generally severe, nor is pain a marked symptom, but heat and a feeling of a foreign body in the eye are usually present. The secretion varies from mucous to muco-purulent, and is mixed with abundant tears. The soaking of the lids in this secretion often causes Blepharitis. Both eyes are almost always affected, though not necessarily simultaneously, and the infectious nature of the disease is constantly seen in the extern departments of hospitals—specially children's hospitals—where whole batches of children will arrive together from one street, or, perhaps, a mother and three or four children, all

suffering from catarrhal conjunctivitis. If a child is seen suffering from this affection in one eye only, with a history of several days' duration, and no other cases in the house or neighbourhood, it is highly probable that careful examination—under an anæsthetic if necessary—will reveal a foreign body under the lid. Often this turns out to be a bristle from a hair brush, though how it gets there is most mysterious.

Causes—Cold is often the only apparent cause, such as sitting by the open window in a railway carriage. In a large proportion of cases, however, there is a clear history of contagion. In other cases it accompanies or follows specific fevers. Epidemics of Acute Conjunctivitis are most common in spring and autumn. A specific micro-organism is said to have been found, but this can hardly be said to have been proved as yet.

Diagnosis—There is not so much danger of mistaking Catarrhal Conjunctivitis for some other affection, as there is of mistaking either of two other affections for it—namely, Purulent Conjunctivitis and Iritis. The former may be quite impossible to distinguish in the early stage, but the swelling of the lids and conjunctiva which rapidly ensue, and the abundant purulent secretion, will generally soon show the real nature of the affection. For the diagnostic points of Iritis, *vide infra*.

Treatment—Early in the course of a severe catarrh of the conjunctiva, cold compresses afford great relief, and may to some extent mitigate its severity. They may be used every two or three hours, for fifteen to thirty minutes at a time. Where ice can be had a large block should be ordered, and small pieces of lint spread over it, one being taken off at a time, applied to the eye for a few minutes, and when it becomes warm replaced on the ice and a fresh one substituted. As a routine treatment in every case,

some mild antiseptic lotion should be ordered, such as Acid. Boric., gr. xv— $\text{ʒi}$ , which may be used freely and frequently, diluted with an equal bulk of hot or cold water—whichever is most grateful to the patient. Mr. Swanzy recommends the following drops, which I have found most useful in severe cases:— $\mathcal{R}$ . Acid. Borici, gr. iv; Zinc. Sulphat., gr. ii; Tinct. Opii,  $\text{ʒi}$ ; Aqu., ad  $\text{ʒi}$ . One drop in the eye morning and evening. When the more acute symptoms are passing off, a ten grain to the ounce solution of Nitrate of Silver may be brushed on the everted lids, all excess of solution being neutralised by freely swabbing with salt and water a few seconds afterwards. This treatment is specially useful in cases which show a tendency to become chronic. Vaseline should be smeared on the lids at night to prevent their sticking together in the morning.

(c) **Follicular Conjunctivitis**—A catarrhal conjunctivitis, either acute or chronic, characterised by the presence in the conjunctiva of rows of minute follicles or granules about the size of pins' heads. These follicles, though microscopically somewhat similar to those found in granular ophthalmia, are clinically quite distinct, as they disappear without leaving any traces behind them. They occur in the palpebral conjunctiva, in rows parallel and close to the margin of the lid. Their significance lies in the fact that where they are present the case is likely to be exceedingly tedious.

*Causes*—No specific virus for this variety of conjunctivitis is known, but it is specially apt to occur in boys and girls in boarding schools; sometimes affecting nearly every scholar in the school.

*Treatment*—The treatment is the same as for the other forms of conjunctival catarrh, according as the affection is acute or chronic. If the follicles remain when the other

symptoms pass off, as is generally the case, an ointment containing five to ten grains Acetate of Lead, or Sulphate of Copper, to the ounce of Vaseline should be well rubbed into the conjunctival sac daily, provided no corneal ulceration is present.

**II. Purulent Conjunctivitis** (Acute Blennorrhœa, Purulent Ophthalmia)—An acute inflammation of the conjunctiva, characterised by great swelling and chemosis of that membrane and the lids, secretion of pus, and presence in the pus of a specific micro-organism—the gonococcus. The two forms in which the affection occurs being so distinct, and both being so important, we shall consider them separately.

(a) Ophthalmia Neonatorum is a form of purulent conjunctivitis occurring in new-born children, due to infection with the specific virus during or soon after birth. Its importance can hardly be exaggerated, when we remember that by far the greatest number of all cases of blindness in the United Kingdom are due to this disease; and, furthermore, that when proper prophylactic measures are taken the disease does not occur, and even where these have been omitted early and careful treatment is always successful.

Cause—The cause is always infection with a vaginal discharge, either during birth or conveyed to the eyes afterwards by the nurse or mother. This discharge need not necessarily be gonorrhœic, for the gonococcus, always present in gonorrhœal discharge, is sometimes present in non-gonorrhœal discharges also. It has been proved that no discharge will produce Ophthalmia Neonatorum unless the gonococcus is present in it. A capital account of the gonococcus, and an interesting discussion concerning the part it plays, will be found in the large manual on "Diseases of the Eye," by Mr. G. A. Berry, of Edinburgh.

*Course*—If infection has taken place directly, the disease usually appears on the second or third day ; if indirectly, it may be a day or two later, both eyes being almost always affected. The preliminary stage, which lasts from one to two days, is characterised by swelling and chemosis of the lids and conjunctiva, and a profuse watery discharge. In mild cases only the palpebral conjunctiva is affected, and the swelling may be slight ; while in severe cases the lids may become exceedingly hard and tense, and of a dark red colour. The next stage sets in with the appearance of pus, and with it the lids become less tense and more easily everted, when the conjunctiva is found to be soft and velvety. The flow varies with the severity of the case, being sometimes very slight. In ordinary cases this stage lasts about six weeks ; the purulent secretion lasting after all swelling and chemosis have disappeared. It is in the early days of this stage that corneal complications are specially liable to occur, and must be warded against, as it is these which form the great danger of the disease. The cornea seldom suffers from interference with its nutrition, as takes place in Gonorrhœal Ophthalmia, but from direct infection, being constantly bathed in pus if the case is not properly treated. Great care is needed in examination, not to scratch or erode the corneal epithelium ; for if this is done, an ulcer is sure to form. Should an ulcer form, it may lead to any of the results described later in the chapter on the cornea.

*Treatment*—Only too frequently the case is treated at home for a week or two by “neighbour women,” with a series of specifics as disgusting as they are futile, and when brought for proper treatment the cornea may be lost by ulceration. When the child is seen it is well to be careful in examining it, for sometimes the lids are glued together with an accumulation of pus behind them, which spurts

up into the eye of the medical man if he stoops over the child's face as he gently draws its lids apart. When the eye is opened, and the pus wiped away with lint or plugs of wool (which should be at once burned), the lids must be drawn apart and the cornea examined. This can often only be done with a lid retractor of some sort. If the cornea is sound, the prognosis is good, and even when it is partially infiltrated it may come all right if kept from further infection. Treatment consists in two procedures—one to be carried out by the medical attendant, the other at home. As soon as free secretion of pus sets in, the lids should be everted daily and brushed with a solution of Nitrate of Silver, gr. x or xx— $\frac{3}{4}$ i, or touched with mitigated stick, the rule being: *the freer the secretion, the stronger the caustic*. In addition to this treatment, an antiseptic lotion must be used at home—the earlier this is begun the better, as the prevention of corneal complications depends chiefly on efficient cleansing. The best lotion is Hydrarg. Perchlor, 1 in 2000, to be mixed with an equal bulk of hot water for use. The eyes should be thoroughly cleansed with this lotion, at first every two hours, night and day, and then at longer intervals, as the secretion becomes less, till it finally ceases.

*Prophylaxis*—It has been proved by Dr. Meyer, of Paris, that septic matter dropped into the conjunctival sac will produce no bad effect if the sac is thoroughly cleansed with an antiseptic lotion soon after. This explains the ground of our treatment for the prevention of Ophthalmia Neonatorum, which consists in applying a strong antiseptic to the eyes of the infant as soon as possible after birth. The best method is the original one of Credé—dropping into the conjunctival sac a solution of Nitrate of Silver, gr. 10— $\frac{3}{4}$ i, and this should be adopted as a routine practice in all midwifery cases.

(b) Gonorrhoeal Ophthalmia is a form of purulent conjunctivitis occurring in adults, due to infection with the specific virus—the gonococcus—either carried directly from the genital organs, or from another eye suffering from this affection. This is one of the most severe and dangerous inflammations of the eye, and may destroy vision in spite of all efforts to save it. It comes on sometimes only a few hours after inoculation, and sometimes in a day or two. The stages are the same as in Ophthalmia Neonatorum, but generally much more severe. In the first stage the early presence of swelling and chemosis distinguish it from a simple catarrh. The upper lid soon becomes so hard and swollen that it covers the lower, and is quite impossible to raise. When the second stage comes on, the lids become softer, and the eye may be better examined. The conjunctiva will be found chemosed, and often overlapping the edges of the cornea. This swelling of the conjunctiva constitutes the great danger of the affection, for it may strangulate the vessels which supply the cornea, in which case the cornea sloughs. If we find the cornea opaque and steamy looking, sloughing is imminent. Should the cornea escape this danger, it runs the risk of ulceration later on from septic inoculation, as in Ophthalmia Neonatorum. During the second stage there is generally abundant purulent secretion, with subjective symptoms as in simple catarrh, but of greater severity. Pain is often marked, and there may be considerable fever.

*Treatment*—The treatment runs on the same lines as that of the infantile affection, namely—free use of anti-septic lotions, and the application of a caustic to the lids when purulent secretion is abundant. If the conjunctiva becomes greatly swollen and overlaps the cornea, and signs of infiltration begin to appear in the latter, a number

of superficial incisions should be made with scalpel or scissors in the conjunctiva, in lines radiating from the cornea, to relieve the chemosis. If, as is generally the case, one eye only is affected at first, every effort must be made to prevent the other becoming affected. The sound eye should be protected by a Buller's shield—a watch glass fastened over the eye with sticking plaster, which allows the use of the eye, but protects it from chance inoculation with the secretion of the other eye. The plaster must not be fastened all round, but a small space must be left at the lower and outer angle for ventilation and the escape of moisture. As an extra precaution we may bathe the eye occasionally with perchloride lotion, doing so freely and frequently if the slightest sign of inflammation should appear.

**III. Croupous Conjunctivitis** is an acute inflammation of the conjunctiva, characterised by all the signs and symptoms of a severe catarrhal conjunctivitis, and in addition, the formation on the palpebral conjunctiva of a greyish white membrane. This membrane does not form till the catarrh has reached its height several days after its onset, and is easily peeled off, leaving the conjunctiva intact, or nearly so. It disappears in a week or two, leaving the eye uninjured. It is wholly a disease of childhood.

*Cause*—As far as is known this is acute catarrh, the membrane being due to the coagulation of the secretion, and having no relation whatever to croup.

*Treatment*—While the membrane is present, only anti-septic lotions must be used ; but when it disappears, and secretion is free, caustics are indicated.

**IV. Diphtheritic Conjunctivitis**—An acute purulent inflammation of the conjunctiva, due to a specific virus, and characterised by a profuse exudation, which forms a true diphtheritic membrane. This is the most dangerous

of all conjunctival inflammations, but is happily very rare in the United Kingdom, though common in some parts of the Continent.

*Course*—After a short period of incubation the lids become swollen, tense, and painful, and a watery secretion is seen. On examining the conjunctiva it is found, in milder cases, red and swollen, with patches of exudation on the palpebral portion ; while in more severe cases the whole conjunctiva may be covered. Diphtheritic patches may be found at the angles of the mouth or in the nostrils, and there are all the general symptoms of diphtheria. In a week, more or less, the exudation begins to disappear, either by being absorbed or by the parts covered with it sloughing away. The purulent secretion is now abundant. As it lessens the cicatricial stage begins, when the parts from which sloughs have separated heal over, often forming adhesions between the lids and the bulb (symblepharon). The great danger in this disease lies in corneal complications. When the exudation is severe the cornea is always destroyed ; when slight, it may be uninjured, and between the two extremes it may be more or less injured by ulceration. A fatal result may ensue from spreading of the disease to the throat or nose.

*Cause*—Always contagion, probably Löffler's "Bacillus Diphtheriae." It rarely affects adults, and then only slightly, being most frequent and most severe in young children.

*Treatment*—All that can be done in the first stage is to apply antiseptic lotions. Caustics must on no account be used till exudation has entirely disappeared. If one eye only is attacked the other should be protected as in Gonorrhœal Ophthalmia. The general treatment is in all respects that of ordinary diphtheria. The effect of the Antitoxin treatment on this form of the disease has not

yet been reported on, but no doubt it will be as effective as in the laryngeal form, if used early.

**V. Granular Conjunctivitis** (Granular Ophthalmia. Trachoma)—An inflammation of the conjunctiva, almost certainly due to a specific virus, characterised by the signs and symptoms of simple conjunctivitis, with the presence of certain bodies known as "granules" in the conjunctiva. These are small greyish-yellow bodies, about the size of a pin's head, found in the fold of conjunctiva between the lid and eyeball, chiefly in the upper lid. They are seen on everting the lid, and telling the patient to look down, so that the fornix is brought well into view. They are scattered about irregularly, and are often likened to grains of boiled sago, under the conjunctiva, and showing through it. They are composed chiefly of lymph cells, with a little connective tissue. Besides these new formations, the lid is altered in appearance by hypertrophy of the papillae, giving it a velvety look.

*Course*—Rarely this disease comes on in an acute form, with marked inflammation and swelling for a week or two. Almost always it is insidious in its onset, and exceedingly chronic in its course. The granulations appear first, and the inflammatory symptoms follow. These gradually increase to a maximum, and then as gradually decrease till they disappear, the whole course generally taking some years; the greater the severity at the maximum point, the longer being the course. The danger lies not directly in the affection itself, but in its complications and consequences.

*Complications*—Besides the risks to the cornea from the purulent inflammation, two complications are liable to arise in the chronic course of the disease, together or separately. One is ulceration of the cornea, the other a peculiar affection of the cornea known as Pannus. This

is a new formation on the surface of the cornea, consisting of hard opaque tissue, with numerous blood vessels, which begins at the upper edge of the cornea, and gradually spreads down till it covers the upper half, or rarely the whole. It may entirely disappear eventually, or may leave patches of opacity.

*Consequences*—In all but the very mildest cases cicatricial contraction follows the disappearance of the granulations, and this leads to most troublesome changes. The commonest of these are trichiasis and entropion, but every variety of deformity may occur in the lids. All degrees of corneal opacity are also found after trachoma, varying from a slight opacity, due to a small superficial ulcer, up to a complete transformation of the cornea into opaque connective tissue, as the result of a long standing pannus.

*Cause*—The origin is always in contagion from another case—probably by a micrococcus. It is found specially where people are crowded together in sleeping apartments, as in schools, barracks, workhouses, &c. It is promoted by insanitary surroundings. It is very common in the South and West of Ireland, but has, happily, almost disappeared in Ulster, where the people live under better hygienic conditions.

*Treatment*—In chronic cases the best treatment is the application of solid Sulphate of Copper to the conjunctiva daily, followed by free washing with water. As alternative methods swabbing the lids with 1 in 500 Perchloride solution, or 10 gr. to the ounce Nitrate of Silver solution, followed by free washing, may be tried. Where more acute symptoms are present, with free secretion, the Nitrate of Silver solution should be brushed on the everted lids daily, and excess well washed off. Ulcers of the cornea are treated by the usual methods—*vide* infra. When pannus is present, weak Atropine drops should be

used, to combat the tendency to Iritis which accompanies it. Bandaging should be avoided. Pannus usually disappears of itself, if the causative affection is treated while it is recent. When, however, it is of old standing, and covers the entire cornea, a special method of treatment is sometimes adopted, which is heroic, but often effective. It is founded on the fact that pannus is lessened by acute exacerbations in the course of the granular ophthalmia, and consists in producing a purulent ophthalmia by brushing the conjunctiva with a 4% infusion of jaquerity bean. This is done three times a day for a day or two, when it produces a severe croupous conjunctivitis, which, when it passes off, leaves the cornea much clearer than before. The most recent treatment for trachoma, said to be very effective, consists in squeezing out each granulation with specially designed roller forceps. Each blade has a stirrup-shaped extremity, the footplate of the stirrup being formed by a small roller—the two rollers meeting when the blades are closed. The lid is everted and brushed with cocaine, and each little granule caught between the rollers and thoroughly squeezed out.

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Having considered the various inflammations of the Conjunctiva, it remains to note briefly other affections of this membrane.

**Pterygium** is a triangular fold of thickened and vascular conjunctiva, extending from over the sclerotic to the cornea, with its apex on the latter. It occurs at the inner or outer side—generally the inner. It may progress slowly for months or years, reaching or passing the centre of the cornea. Finally, the vessels disappear and it becomes stationary.

*Cause*—It arises from mechanical irritation of the cornea, and is generally seen only in old people who have been much exposed to dust or wind.

*Treatment*—Unless it interferes with vision, or is steadily progressing, it had better not be interfered with. If we decide to remove it, it is grasped with toothed forceps and carefully dissected off, and the conjunctiva sutured.

**Dermoid Tumours** are very rare, and always congenital. They are found oftenest just on the margin of the cornea, at the temporal side. They are solid, flat, pale-red or white, with a surface like ordinary skin. If large, they may be removed like pterygium.

**Lipomata**, or fatty tumours, are also rare. They are found at the upper temporal side, and show as a small yellow mass through the conjunctiva. They are congenital, but may increase at puberty, and can be removed if necessary.

**Malignant Tumours** of the conjunctiva are rare. Epithelioma and Sarcoma occur. The former is pale, flat, broad-based, and extends slowly over the surface of the eye, generally ulcerating. Sarcoma is pigmented, grows in height, and has a distinct pedicle. Enucleation is the only treatment.

**Sub-Conjunctival Ecchymosis** is frequently seen, after blows on the eye, in severe forms of Conjunctivitis, and occasionally in whooping cough. If found *far back* on the eyeball, after an injury, such as a fall on the head, it aids in establishing a diagnosis of fracture of the base of the skull. When seen in old people, without obvious cause, it indicates weakness of the vessels, and warns us of the possible occurrence of cerebral hæmorrhage.

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### CHAPTER III.

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#### **PHLYCTENULAR OPHTHALMIA**

(Strumous or Phlyctenular Conjunctivitis and Keratitis, Conjunctivitis Lymphatica).

This is one of the most important, as it is the commonest, of all superficial diseases of the eye. I find that in a hundred consecutive cases of eye disease in my Clinic at the Ulster Children's Hospital, no less than forty cases were of this affection, or its Sequelae.

Phlyctenular Ophthalmia is a focal inflammation of the conjunctiva, corneal margin, or cornea, characterised by the eruption of one or more nodules of exudation under the epithelial surface. The nodules consist of little collections of lymph cells, and they vary in size from that of a pin head down. They do not always remain stationary, but often show a tendency to move towards the centre of the cornea. Soon after formation the little nodule comes to a point and breaks down, leaving a small grey ulcer, with the surrounding tissue injected. If on the conjunctiva, they may give comparatively little trouble, but on the cornea they give rise to photophobia, often intense. Indeed, a glimpse at a small patient, with his face buried in his hands, or on his mother's shoulder, avoiding all light and air, is almost sufficient to establish a diagnosis. If the child cannot be got to open his eyes, he must be laid on his mother's lap, with his head resting on the surgeon's knees. The lids are wiped dry, and then, the head being held firmly between the knees, the lids are separated with the fingers, or a lid retractor if necessary, and the eye examined. In some of the worst cases of

photophobia, no grey spot is seen, but on careful examination we find at some part of the corneal margin little rough grains, like sand, with injection in the neighbouring conjunctiva. These are miliary phlyctenules. The photophobia is due to irritation of the branches of the fifth nerve in the cornea, which arises from the fact that the lymph cells in travelling from the margin of the cornea to the nodule of exudation follow the tracks of the nerves. It is not relieved in the dark, but is relieved by cocaine or atropine applied to the cornea.

*Course*—The phlyctenule, as we have said, soon breaks down into a little ulcer. This may remain where it is, or may move towards the centre of the cornea, carrying a leash of vessels in with it from the margin. If it heals, it leaves an opacity in its whole course. It may get deeper and perforate (see under Ulcers of Cornea). The miliary phlyctenules by breaking down may form a long marginal or ring ulcer—a dangerous form, as it interferes with the nutrition of the cornea. This affection generally runs on for months, or even years, as hospital patients are only too liable to stop treatment as soon as the more acute symptoms pass off. When it runs a long course, complications are likely to arise. The intense photophobia may cause Spastic Entropion, and the constant soaking of the lids in tears causes Ophthalmia Tarsi.

*Cause*—No specific virus has been recognised, nor does it seem likely that such exists. The immense majority of cases occur among the children of the poor, who live in damp, bad air, are dirty, and are badly fed—not only as regards their food supply, but the manner in which it is used. The few cases occurring in the better classes generally follow some weakening disease, such as measles; and when adults suffer, it is only as a recrudescence of the affection they have suffered from in childhood. Such a

large proportion of cases show signs of strumous disease, that this is often called Strumous Ophthalmia.

*Treatment*—This must be local and general. Three procedures are carried out in local treatment, according to the symptoms—(a) Great photophobia indicates atropine—best used as drops with some antiseptic, such as  $\mathcal{R}$ . Atrop. Sulph., gr. 2 ; Acid. Boric., gr. 8 ; Aquae,  $\mathfrak{z}\text{i}$ . Frequent cold sponging of the face and neck tends to relieve photophobia. (b) Early in the disease calomel should be dusted into the eye daily, unless a corneal ulcer is present. (c) Later on, when the inflammatory symptoms are less, yellow ointment (Hydrarg. Ox. Flav., gr. 6 ; Vaseline,  $\mathfrak{z}\text{i}$ ) should be used twice daily at home, or a stronger ointment once daily applied by the surgeon. A little piece is to be inserted in the conjunctival sac, and the lids gently rubbed, with a circular motion, so as to bring it into contact with all parts of the cornea.

*General Treatment* consists in the administration of cod-liver oil and iron, and the improvement of hygienic surroundings as far as possible. Special instruction should be given about regular meals—the general plan of the poor being to feed their children the whole day long. To town children, a change to the country, and specially to the seaside, is often of the greatest service. Directions must also be given that no bandage or handkerchief is to be tied over the eyes, but a wide-brimmed hat or a stiff shade, standing well out from the forehead, should be worn to protect the eyes from bright light.

*Sequelae*—Ulcers of the cornea constantly result from Phlyctenular Ophthalmia ; indeed, when a large single phlyctenule is found on the cornea, its breaking down and forming an ulcer may be taken as the normal course of events. Such ulcers must be treated by the ordinary methods—see next chapter. The most troublesome and

serious result of Phlyctenular Ophthalmia is the opacity which it so often leaves in the cornea. We see many cases where, year after year, a child suffers every winter, each attack leaving the cornea a little more opaque than before. A single small opacity is not likely to interfere much with vision, unless in the centre of the cornea, but much injury to vision is occasioned by the diffuse cloudy opacity commonly found after repeated attacks of Phlyctenular Ophthalmia—specially as in these repeated attacks both eyes are sure to be affected. Consequently, every means should be used to promote the absorption of the opaque tissue, and to this end nothing seems so good as the treatment advocated by Professor Reuss, of Vienna—regular massage of the eyeball for several minutes, night and morning, with a weak mercurial ointment. My practice is to order Ung. Hydrarg. Ox. Flav., gr, vi—ʒi, and tell the patient's parents to put a small piece of this inside the lower lid, then rub the finger with a gentle circular motion over the closed lid for two or three minutes. Children can soon be taught to do this themselves, and the process should be repeated night and morning, and kept up for at least three months. It might seem hopeless to get ignorant people to take so much trouble, but I have found parents in the working classes quick to appreciate the fact that "a scum over the sight" will be a great hindrance to wage earning as the child grows up, and consequently willing to take great trouble to remove it. Where a dense opacity is situated over the pupil, vision may be improved by an iridectomy, to form an artificial pupil. A slight opacity is sometimes called a nebula ; a dense one, a leucoma.

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## CHAPTER IV.

## DISEASES OF THE CORNEA.

**Keratitis**—Inflammation of the Cornea may be suppurative or non-suppurative, and local or general. In all cases where an affection of the cornea is known or suspected, a careful examination is to be made, three points being noticed—*first*, its transparency; *second*, the character of its surface; and, *third*, the shape or form of its surface. The transparency is interfered with whenever there is exudation in or upon it, *i.e.*, whenever it is inflamed in any degree. The reflecting power of the surface is altered when the cornea is inflamed, and it becomes dull or steamy looking, like ground glass. Its shape is altered by any loss of substance on its surface.

**Suppurative Keratitis** includes (*a*) Abscess, (*b*) Ulcer, both being characterised by the formation of pus, and more or less opacity following.

(*a*) **Abscess** of the cornea consists in a local infiltration of pus in its deep layers, with clear cornea before and behind. It appears as a more or less dense yellowish-white opacity at or near the centre, the surface being dull over it. It is accompanied by iritis, generally severe, and turbidity of the aqueous humour. Pus appears in the anterior chamber, settling down to its lowest point: this is known as hypopyon. There is swelling of the lids, injection of the conjunctiva, and intense neuralgic pain. Soon the abscess bursts anteriorly, forming an ulcer, when the acute symptoms often disappear—or they may go on to complete destruction of the cornea, or even the eyeball.

**Cause**—The cause is probably always infection, either

from within or without. The former occurs in acute infectious diseases, such as smallpox, when it generally destroys vision. The latter necessitates some break in the epithelial surface, and is generally associated either with some chronic affection of the conjunctiva or lids or with a low state of health, and is seen chiefly in adults. According to Fuchs it is commonest in very hot weather; but in Ulster, at any rate, it is most frequently seen in winter, in elderly ill nourished men who have been much exposed to the weather.

*Treatment* consists in the use of atropine and iodoform, with bandaging of the eye, and frequent hot stuping to relieve the pain. If the case be severe, the sooner the abscess is opened by a free incision from side to side the better; or better still, it may be opened with the cautery.

(b) Ulcer of the Cornea develops from the breaking down of a local infiltration near the surface of the cornea. The ulcer often remains surrounded by grey infiltration, and grows in size by the breaking down of fresh portions of tissue; or it may remain small, but increase in depth. If it extends, the chief danger lies in the great opacity which follows it. If it deepens, it may perforate.

*Varieties*—Many varieties of ulcer are recognised, of which the following are the chief:—

PROGRESSIVE ULCER is recognised by the loss of substance on the cornea, by its rough edges, and by its dull ground-glass floor, showing that the epithelium has not grown over it. It is accompanied by the signs of irritation described in the last chapter—pain, photophobia, &c. Some iritis may also be present in severe cases.

REGRESSIVE ULCER is recognised by the disappearance of acute symptoms, and by the little hollowed out cup where the loss of substance took place, being lined with new epithelium, which reflects light as the general healthy

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surface does. Infiltration, if it was present, has disappeared.

SERPIGENOUS ULCER may be compared to a little wave flowing slowing over the cornea—a raised up wave of infiltration being followed by a hollowed trough where there is loss of substance. The wave slowly progresses in one direction, leaving a track of opacity in its wake. It is a severe form, liable to damage the eye seriously.

MARGINAL RING-SHAPED ULCER extends like a snail track round the cornea, sometimes even the whole circumference being affected, and nutrition cut off from the centre. There is not much infiltration. The risk lies in interference with the nutrition of the cornea, and consequent sloughing.

Causes—Ulcers of the cornea are almost never primary, except when traumatic. In the great majority of cases, ulcers arise as complications in conjunctival affections—*vide* Chapter II., or follow Phlyctenular Ophthalmia.

Treatment—Conjunctival disease must be treated if present. If caustics are used, they must not be allowed to touch the cornea. In progressive ulcers the eye should be bandaged, unless there is a very large quantity of secretion, which a bandage would only serve to retain. Atropine drops (gr. ii— $\frac{3}{4}$ i) should be instilled daily, to check any tendency to Iritis, except when a marginal ulcer becomes deep, and there is a risk of perforation, when Eserin drops (gr. i— $\frac{3}{4}$ i) should be used to contract the pupil and lessen the chances of prolapse of the iris. When a large ulcer is present, accompanied by much infiltration, and perhaps hypopyon, iodoform should be dusted on the eye, and hot stuping used. If steadily progressing over the cornea, the infiltrated edge should be cauterised. This is best done with the special galvano-cautery for ophthalmic use, or Pacquelin's thermo-cautery, but may be done with a probe heated in a spirit lamp. If the ulcer becomes deep,

and perforation threatens, it should be brought about artificially, the floor of the ulcer being perforated with a fine knife. By so doing the aqueous can be allowed to escape slowly, and the iris may be undisturbed, whereas if perforation takes place naturally the aqueous gushes out and probably carries the iris into the wound, causing a prolapse. It is seldom possible to permanently replace the iris if once prolapsed, and if the attempt to do so fails, the eye must be cocaineised, and the prolapse snipped off with scissors when fresh. If not seen for two or three days it should not be touched, but left to heal in the perforation, only being kept clean with antiseptic lotions. In small perforations the iris may not prolapse, but simply become attached to the wound, forming an adherent leucoma. When an ulcer is healing, and epithelium has formed over it, unguentum flavum (see p. 24) should be used, stopping it if there is any return of acute symptoms. The density of opacity which follows an ulcer depends largely on its depth: if superficial, the opacity may be slight, and ultimately disappear under treatment; but if deep, a dense white leucoma follows, which can never be removed. Operations have been tried, but hitherto without success.

**Non-Suppurative Keratitis** includes two superficial and three deep forms—(a) Pannus, (b) Herpes Corneae, (c) Interstitial Keratitis, (d) Punctate Keratitis, (e) Sclerotising opacity of the Cornea.

(a) **Pannus** has been considered with Trachoma, p. 18

(b) **Herpes Corneae**—Small clear vesicles occur on the cornea in two forms—*first*, in large numbers, with great signs of irritation, and accompanied by similar vesicles on the face, ears, &c., in febrile diseases—most often in influenza; *second*, true herpes zoster, in which the cornea is more deeply, and often permanently, affected. In both

varieties treatment consists in the use of atropine and bandaging.

(c) **Interstitial Keratitis** (Parenchymatous Keratitis) is a chronic inflammation of the cornea, occurring generally in youth, characterised by a diffuse cloudy infiltration of its substance, and a dulness and loss of lustre on its surface.

*Course*—A typical case runs somewhat as follows :—The patient—generally between six and eighteen—is found to have a dull grey opacity at one part of the cornea, oftenest near the margin, with some inflammatory symptoms in the neighbouring conjunctiva, not severe. The opacity is diffuse, and shades off imperceptibly into the surrounding healthy tissue. It gradually spreads till in about two months it has reached its height, covering the whole cornea, so that the iris cannot be seen. The surface of the cornea is dull and steamy looking, and may show slight elevations, in which case the opacity will be seen to be mottled, being denser in some spots than in others. Soon the inflammatory signs abate, and the cornea begins to clear from the circumference to the centre, the clearing not being complete till about six months after the onset of the disease. Meantime the second eye is almost always affected, seldom simultaneously, but generally from a few weeks to several months after the first, and runs a similar course. This is a picture of an ordinary severe case, but much lighter cases are seen, as well as some more serious in which the iris is affected.

*Diagnosis*—The recognition of this disease depends on its slow onset and course, the deep position of the infiltration, and the total absence of ulceration.

*Cause*—In the great majority of cases Interstitial Keratitis is an evidence of inherited syphilis. Signs of this will generally be found in the peculiar flat face and low-bridged nose, scars at the angles of the mouth, showing

old ulceration, and peg-shaped, irregular, and notched incisor teeth. The family history, where obtainable, may help to confirm our diagnosis. Where no signs of specific disease can be found, signs of scrofula may occur, but in some cases no cause can be traced, though the more carefully we search for syphilis the fewer will these be.

*Treatment*—Constitutional treatment must be carried out in all cases. Mercury should be cautiously given in severe cases, no matter what the history, and iodide of potash in less severe cases, with cod-liver oil and iron. Locally atropine should be used till signs of clearing begin to appear, when unguentum flavum should be used, at first only by the medical man, watching to see that it does not cause any return of the inflammatory symptoms. The eyes must not be bandaged, but a shade of some sort worn to moderate the light. The patient or his friends should be warned of the chronic nature of the affection, and of the practical certainty of the second eye being affected, if it is not so already.

(d) **Punctate Keratitis** (Descemetitis)—An inflammatory affection of the lining membrane of the cornea—the membrane of Descemet—characterised by slight opacity of the cornea and a deposit on its posterior surface of small brownish-grey nodules, almost microscopic in size. They are generally seen in the lower quadrant of the cornea, scattered over a roughly triangular area. This affection is oftenest seen with a chronic iritis or iridocyclitis, and has been generally looked on as a complication of either of the latter; but Professor Snellen has lately studied the pathology of it, and declares that the little nodules are masses of short bacilli, which stain in carbol-fuchsin; that it is a disease *sui generis*; that the affection of the uveal tract is secondary to it, due to irritation by the toxins which the bacilli produce; and

that under the internal administration of Sodium Salicylate this irritation disappears.

(e) **Sclerotising Opacity of the Cornea** is found as a complication in chronic inflammation of the sclerotic. The margin of the cornea slowly becomes opaque, till finally a dense white opacity is seen extending two or three millimetres into the cornea, apparently a continuation of the sclerotic. Treatment is that of Scleritis.

**Staphyloma of the Cornea** consists in a bulging cicatrix, following perforation and a prolapse of the iris. It is called total if the whole cornea bulges out, partial if only a portion of it. The protruding part may vary in size from a mere slight elevation to a staphyloma nearly as large as the eyeball.

*Treatment*—Prophylaxis is most important. Ulcers of the cornea should be kept under observation a long time, and if the scar shows any tendency to bulge, an iridectomy should be performed. If a staphyloma has formed, and it is small, a cure may sometimes be effected by incising it and bandaging the eye for a length of time. In more severe cases the protruding part may be cut off and the edges of the wound sutured together. In other cases, again, nothing but an enucleation can be done, the staphyloma is so large.

**Conical Cornea** is a rare condition in which the cornea loses its normal spherical shape and approaches the conical. There is disturbance of vision which cannot be satisfactorily corrected by glasses, and when advanced the conical shape can be recognised on looking at the eye from the side. It generally affects both eyes. Treatment is unsatisfactory, but eserine drops may be used to diminish intraocular tension.

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## CHAPTER V.

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DISEASES OF THE SCLERA AND IRIS,  
AND GLAUCOMA.

**Inflammation of the Sclerotic** is not common. It is usually confined to the anterior part, and may be superficial or deep.

SUPERFICIAL SCLERITIS (Episcleritis) is a local inflammation, characterised by the presence of a small nodule of exudation in the sclera. This nodule is elevated, generally flat-topped and surrounded by inflamed tissue of a violet colour. It can not be moved on the sclera, as a phlyctenule can. Pain is sometimes present, but not always. After a few weeks the inflammation lessens, and in the course of time the nodule disappears, only to be followed by another at some other spot; each nodule leaves behind it a slight dark discolouration of the sclerotic. This process runs on for years, but finally ceases without affecting the sight. It occurs only in adults, and seems to be frequently connected with a rheumatic or a gouty diathesis. Treatment should be directed against this if present, and locally consists in atropine and hot stuping if there is pain, and massage, with a weak mercurial ointment, as inflammatory symptoms abate.

DEEP SCLERITIS is also a local inflammation, characterised by swelling, which is less sharply defined, and is more like a bulging of part of the sclera, and the inflammation tends to spread to the cornea and ciliary region. The inflamed portion of the sclera is a deep purple-red colour. The inflammation usually runs on for years, unaffected by

treatment, and affecting both eyes, till it finally destroys vision, or at least seriously injures it, by causing opacity of the cornea, iritis, choroiditis, &c. It is found in young people, specially girls, and is associated with tuberculosis, scrofula, &c., against which treatment should be directed.

**Iritis and Cyclitis**—The iris and ciliary body are so closely connected that if one is inflamed the other is generally more or less affected. The ciliary body, however, is not accessible to examination, like the iris, and it is only by the presence of certain symptoms that we recognise a cyclitis as present with an iritis; and, moreover, the treatment is the same whether it be present or no, so that for clinical purposes we may consider the iris only. Cyclitis without iritis is very rare, and is only recognised by the presence of exudation without inflammatory symptoms in the iris. Iritis in general presents certain symptoms, varying in degree according to the type of inflammation present.

1. There is more or less irritability of the eye, with photophobia, lachrymation, &c.

2. In acute cases the whole conjunctiva is injected, giving the appearance of a conjunctivitis; and in both acute and chronic there is pericorneal injection. This is seen as a pink zone about two millimetres broad on the sclerotic surrounding the cornea. Where general injection of the conjunctiva is present, the lid should be lightly pressed against the eye with the finger and quickly slipped aside. For a second the conjunctival vessels are empty, and we can see whether the pericorneal vessels are injected or not, as they are not affected by the pressure.

3. The iris is discoloured by hyperaemia and exudation. A blue or grey iris appears greenish, the change being seen on comparing it with the other eye, if it is not affected. A brown iris appears muddy.

4. There is exudation. This may be—(a) Into the iris, altering its colour, and making it thick and heavy looking. (b) Into the anterior chamber, causing a turbidity of the aqueous. (c) Into the posterior chamber; this is specially abundant when there is cyclitis. The effect of this may be to glue the iris at one or more points to the lens capsule (posterior synechiae); or it may glue the whole edge of the iris to the lens (annular posterior synechia); or it may glue the whole iris flat against the lens (total posterior synechia); or it may fill the pupil with lymph (occlusion of the pupil). The effect of annular synechia is to shut off the posterior chamber from the anterior (seclusion of the pupil), and the effect of total synechia is to obliterate the posterior chamber entirely. (d) In irido-cyclitis only; into the vitreous, causing floating opacities.

5. The functions of the iris are interfered with. It reacts to light slowly, and comes to rest in a more contracted state than normal.

6. There is pain—generally a marked symptom. It is of a neuralgic character, so much so that cases of chronic iritis have been treated for neuralgia by careless surgeons who have overlooked the slight pericorneal injection and other eye symptoms. The pain shoots up the side of the nose, the forehead, and temple, becoming much worse at night.

*Varieties*—For clinical purposes iritis may be divided into—(1) Plastic, (2) Serous, and (3) Purulent Iritis.

(1) **Plastic Iritis** is the ordinary simple acute form. The irritability, injection, and pain are all marked. Vision is interfered with by the turbidity of the aqueous. An attack may develop very suddenly, becoming severe in a few hours, and may last days or weeks. Some forms are specially liable to recur. Posterior synechiae are nearly certain to form, unless treatment is begun early. They

are recognised by the irregular shape of the pupil when dilated with atropine.

*Diagnosis*—An acute iritis has to be carefully diagnosed from two other affections—Conjunctivitis and Glaucoma. From the first it is distinguished by the presence of pericorneal injection, turbidity of the aqueous, alteration in colour of the iris, and its immobility, and the severe pain. From glaucoma it is distinguished by the fact that the pupil is contracted, while in glaucoma it is dilated, and though the tension may be slightly raised, the eye is never of the stony hardness found in glaucoma. It is most important to remember these points, as to treat a case of glaucoma as one of iritis simply means to destroy vision.

(2) **Serous Iritis** is almost always complicated by cyclitis. It is a chronic affection, often without any very obvious eye symptoms at first, but pericorneal injection will always be found on careful examination. The pupil may tend to dilation rather than contraction. Pain is sometimes absent, sometimes marked. It runs on for a long time, and is particularly liable to extend to the choroid and retina. If it progresses unchecked, it ultimately destroys useful vision, either by blocking the pupil with lymph or by extending to the back of the eye. It almost always attacks both eyes.

(3) **Purulent Iritis** is characterised by an exudation of pus in the iris itself, which may become enormously swollen, either over its whole surface or at one point. The pus may also be poured into the anterior chamber, forming a hypopyon. Hæmorrhage from the iris sometimes takes place.

*Causes of Iritis*—Syphilis is much the most frequent cause, in its secondary stage, when plastic iritis is a very common occurrence. Next to this come rheumatism,

gout, and gonorrhoeal rheumatism, in all of which plastic iritis may occur. Rheumatic iritis is specially liable to recur again and again. Some few cases of plastic iritis must be put down as ideopathic. The cause of serous iritis is unknown, but it seems to lie in some alteration in the blood. About nine cases out of ten occur in women. Purulent iritis may be traumatic, or may arise from septic embolism, and is sometimes seen in diabetes and acute infectious diseases. Irido-cyclitis arising from sympathetic irritation will be considered in Chapter XII.

*Treatment*—In all acute cases of iritis our mainstay is atropine. It should be used as drops, gr. iv— $\frac{3}{4}$ i at first, and weaker later; a drop in the conjunctival sac three times daily. If adhesions (posterior synechiae) have already formed when we see the case, an attempt should be made to break them down by dropping a drop of atropine into the eye once every five or ten minutes for six times, as Mr. Swanzy suggests. Internally mercury should be given—best as perchloride. To relieve pain and photophobia the patient should be kept in a dark room, and the eye frequently bathed with very hot water, or pads of hot flannel held to it. Leeching at the inner or outer angle of the eye relieves pain greatly, but a better plan is for the surgeon to make a small cut on the temple with a lancet and draw as much blood as he thinks well with a cupping glass. Where rheumatism is present, salicylates should be given. In serous iritis only very weak solutions of atropine should be given. Free action of the skin, brought about by Turkish baths or the subcutaneous injection of pilocarpine, is beneficial, as is also paracentesis of the anterior chamber, and dry cupping on the temple. Change of air and regular exercise should be recommended. In purulent iritis treatment is of little avail, but quinine may be given internally.

**Tumours** of various sorts may occur in the Iris, but none are common. *Cysts* are generally traumatic in origin. *Tubercle* sometimes attacks the iris, appearing as small white growths in its substance. *Malignant disease* is exceedingly rare, except as an extension from other parts. Gummata are occasionally seen in the Iris.

**Coloboma of the Iris** is a congenital absence of part of the iris, making it appear as if an iridectomy had been done.

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## GLAUCOMA.

**Glaucoma** is generally a disease of advanced life, characterised by increase of the intraocular pressure. It may be primary or secondary; but the latter only occurs as a complication in some other eye disease, and is unimportant compared to primary glaucoma, which, on account of its frequently insidious onset, its liability to be mistaken for other affections, its serious consequences, and its amenity to treatment in the early stages, is one of the most important of all eye affections for the practitioner to be familiar with. The increased tension is recognised by telling the patient to look down and close his eyes, the surgeon then laying the tips of his forefingers on the closed lid, about half an inch apart, and pressing lightly with each alternately. He does this on both eyes, and on some other person's eye if necessary, to get an idea of the normal tension. In severe cases of glaucoma the eye feels like a stone. In addition to the increase of tension, certain other symptoms are present, arising from that increase. The cornea appears dull, like glass with steam on it, and as a result of this certain disturbances of vision take place, the patient complaining that when he looks at a light he sees coloured rings round it, and objects are often dim.

The sensibility of the cornea to touch is lowered. The anterior chamber is shallow, and the pupil more or less dilated, and not freely movable. The pupil, instead of appearing black, has a peculiar greenish look, from which the disease takes its name (*γλαυκος*). On ophthalmoscopic examination the optic disc is seen to be cupped, or hollowed out, far beyond what is normal. Instead of the normal disc we may find a white pit, over the edge of which the vessels disappear. Lastly, the field of vision is interfered with, there being generally a concentric diminution on all sides. This may progress till the patient only sees objects directly in front of his eye, when he has the sensation of looking through a tube. Glaucoma presents two types—Congestive and Simple.

CONGESTIVE GLAUCOMA is often called inflammatory, but there is no real inflammation, and the name suggested by Mr. Priestly Smith, of Birmingham, whose researches on glaucoma are of world-wide reputation, is certainly better. There are usually some premonitory symptoms—occasional dimness of vision, rings of light, &c., and weakening of the power of accommodation, necessitating frequent increase in the strength of glasses for near work. An acute attack then comes on, with violent pain in the eye and over the head, lachrymation, injection of the conjunctiva, and sometimes chemosis, diminution or even total loss of vision, and occasionally vomiting. Many attacks present a sub-acute character, with these symptoms in a modified form, and some chronic cases have acute exacerbations. These acute or sub-acute attacks last a few days, and recur again and again, each leaving the vision a little worse than before, till at last it is completely lost.

SIMPLE GLAUCOMA runs a chronic course without any of the marked symptoms that characterise the congestive

form. No change in the pupil is seen at first, and as the tension varies it is only on repeated examination we can be sure that it is raised. Cupping of the optic disc, however, is marked, and upon it and the contraction of the field of vision our diagnosis must often depend. This form of glaucoma is generally far advanced before it comes under treatment, and, as a rule, runs on to complete loss of vision in spite of all efforts to check it.

*Cause*—The origin of glaucoma and its pathology are alike very incompletely known, in spite of all research.

Fig. 1.  
Section of normal eye.

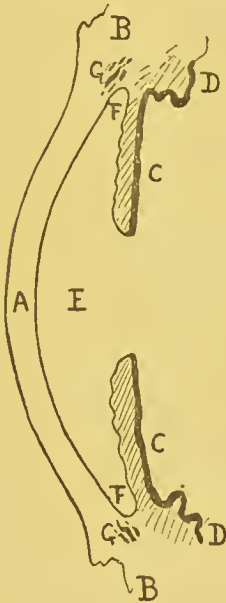
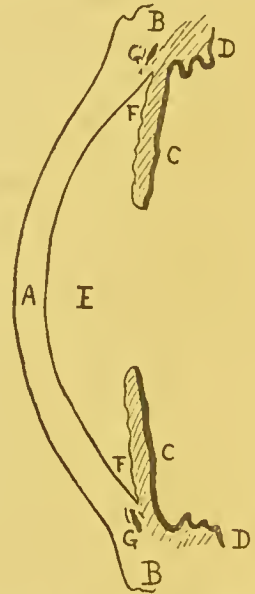


Fig. 2  
Section of glaucomatous eye.



A, Cornea. B, Sclerotic. C, Iris. D, Ciliary body. E, Anterior chamber.  
F, Iris angle. G, Schlemm's canal.

It is essentially a disease of advanced life, patients being generally over fifty. General health seems to have no connection with it. Scores of theories have been advanced to account for it, but all are more or less unsatisfactory.

The least so is that of Mr. Priestly Smith, who attributes the origin of Glaucoma to blocking of the iris angle, or angle between the root of the iris and the cornea (F, fig. 1). This is brought about by the lens pressing forwards, and so pushing the base of the iris against the cornea and obliterating the normal space between them (F, fig. 2). The fact that the lens becomes larger in later life is adduced to account for glaucoma being a disease of advanced life. In the iris angle is a quantity of loose tissue through which fluid is drained from the anterior chamber into Schlemm's canal (G, figs. 1 and 2). When the angle is blocked, fluid cannot escape freely, and intra-ocular tension is increased, leading to all the other signs and symptoms of glaucoma. The accompanying figures are sketches from actual specimens in my possession.

*Treatment*—Eserine may do some good of a temporary nature by reducing the tension, and should always be used in simple glaucoma ; but in acute and sub-acute forms an operation should be done at once, either iridectomy or sclerotomy—preferably the former. The portion of iris removed must be large, and must be taken from the root. If done soon after the onset of an acute attack, though vision is almost completely lost, it is restored by the operation ; but when vision has been lessened for a length of time, little improvement can be hoped for, though the further progress of the disease may be checked. In congestive glaucoma about three-fourths of the cases operated on are improved, and very few made worse, whereas in simple glaucoma less than one-fourth are improved, and almost as many become rapidly worse, consequently the advisability of operating in a case of simple glaucoma is very doubtful. In secondary glaucoma—complicating cataract and other operations—it is often sufficient to do paracentesis of the anterior chamber, allowing the aqueous

to escape slowly. Glaucoma is sometimes caused by the use of atropine in old people's eyes. In this case also a paracentesis is generally sufficient, as it reduces tension till the effects of the atropine pass off.

*Results of Glaucoma*—A series of attacks of congestive glaucoma leads not only to loss of vision but to general disorganisation of the tissues, resulting in lowering of tension and shrinking of the eyeball—phthisis bulbi. Simple glaucoma may become acute, or may run on for years, ending in atrophy of the optic nerve.

## CHAPTER VI.

### DISEASES OF THE LENS.

All changes in the lens come under one of two heads—*first*, changes in character ; *second*, changes in position. Changes in the character of the lens are only recognised by one sign :—loss of its normal transparency, which we call cataract.

**Cataract** may be briefly described as opacity of the lens. It presents a number of varieties, the chief of which are—(1) Senile, (2) Congenital, (3) Anterior polar or Pyramidal, (4) Traumatic.

(1) **Senile Cataract** is a progressive degeneration of the lens, causing loss of its transparency, occurring in persons over forty or forty-five years old. Certain symptoms occur pointing to cataract, the most prominent being disturbance of vision, the character of which depends on the form of the opacity. Sometimes the patient simply complains of

dim sight, not being able to recognise a friend across the street, and so on. Or, he may complain of regular objects—such as the bars on a window—appearing broken up and irregular. The former complaint is due to a general cloudiness of the lens, the latter to the occurrence of sharply defined areas of opacity. It is not uncommon to find also some symptoms of chronic conjunctivitis present. Cataract generally affects both eyes, but not always simultaneously, so that when vision is tested one eye is found to be better than the other. Objective examination is carried out in two ways—first by oblique illumination of the eye, and second by direct examination with the ophthalmoscope. For the first examination, we place the patient so that a good light falls on the right side if we wish to examine the right eye, and on the left for the left eye. This light may be diffuse daylight (*not* sunlight), or lamp or candle light: with a convex lens we focus the light on the pupil, so that any opacity in the lens is lit up and shows as a grey mark against the black background. The light must be as much as possible from *the side*, and not in front of the eye, for in that case the light falls on the retina and the pupil contracts. The second examination is made by looking at the lens directly with the ophthalmoscope—best at six or eight inches from the eye—with a low convex lens. The light should not be at all bright, as this would cause great contraction of the pupil. In this examination opacities of the lens show out black against the red fundus. Senile cataract may commence at the centre of the lens as a diffuse cloudiness (nuclear cataract), or at the periphery of the lens as sharply defined points—striae or spicules—(cortical cataract). The latter do not interfere seriously with sight so long as they do not encroach on the pupillary area.

*Cause and Pathology*—The cause of the degeneration

which brings about opacity of the lens is quite unknown. It occurs in the most perfectly healthy persons as often as in the feeble or delicate, and we can no more tell why one person's lens should become opaque and another's not, than we can tell why one person's hair should become grey and another's remain dark. The immediate cause of the opacity is an irregular shrinking of the lens, which causes the separation of its fibres at some points. The spaces thus formed between the fibres are at first filled with clear fluid, but this fluid soon becomes opaque, and this change is followed by opacity of the surrounding fibres.

*Prognosis*—Considering the early stage at which cataract is often diagnosed, the advanced age at which it is generally found, its slow progress, and the consequent possibility of the patient's not living to lose his sight, it is needless cruelty, in the majority of cases, to tell the patient that he has cataract and will ultimately become blind. It is generally wiser and more humane to tell the patient that "he is not as young as he was, and his sight has failed a little"; but in such cases the matter should always be explained to some relative. If this is not done the medical man may lay himself open to the charge, at some future time, of having failed to recognise the cataract. The patient or his friends having been informed that he has cataract, the first question asked is: "How long will it be before he becomes blind?" To this no definite answer can be given, but certain signs guide us a little. The older the patient, the more likely is the process to advance steadily. Nuclear cataract causes blindness sooner than cortical. Cortical cataract with fine sharply defined spicules, all pointing towards the centre of the lens, is likely to progress slowly. Progress is often quite irregular: a cataract may increase rapidly for some time then remain

stationary, or *vice versa*. In cases where blindness is spoken of as a possibility, the medical man should tell the patient that in the event of sight being lost an operation may be done with every hope of restoring it to a useful degree.

*Treatment*—Operation is the only treatment for cataract—the opaque lens being removed and its function being fulfilled by convex spectacles. As a general rule an operation is better deferred till the cataract is “ripe,” which is recognised by two signs. When examined by oblique illumination the iris casts no shadow on it, showing that the lens is opaque right up to the anterior capsule. The other sign is that the lens appears uniform in opacity, and shows no sectors of a mother-of-pearl appearance. Before deciding on operation, it is necessary that we should make as sure as possible that no disease of the fundus is present. This is done by examining the patient in a dark room with a taper or candle. When the second eye is covered he should be able to see the light of the taper through the cataractous lens when held ten or fifteen feet off, and point at once to it. It must be held above, below, and on each side in turn, so that the light may fall on every part of the retina. Full details of the operation for cataract will be found in larger manuals than this, but the steps are as follows in the ordinary operations :—

1. The eye is cocainised—best with a few grains of Hydrochlorate of Cocaine, and after this has taken effect, a speculum is introduced and the whole eye and conjunctival sac thoroughly cleansed with an antiseptic lotion. ✕

2. The eye is held by fixation forceps, which catch the conjunctiva below the cornea. A Graefe's knife is entered in the corneal margin, with its edge upwards, and carried through the anterior chamber and out at the opposite corneal margin, so as to lie in a horizontal line three milli-

*To be used only when the patient is under the influence of the anæsthetic.*

metres below the highest point of the cornea. The bridge of tissue is then divided by a slow sawing motion, so that the whole incision lies just in the corneal margin.

3. An assistant holds the fixation forceps, the surgeon catches the iris with fine iris forceps, draws it out of the wound, and cuts off with scissors the little piece drawn out—thus doing a small iridectomy.

4. A cystotome (an instrument like a fine bent probe, with a tooth pointing side ways at the end of it) is passed into the wound, and the capsule of the lens opened by lightly scoring its surface with the little tooth—first from side to side, and then up and down.

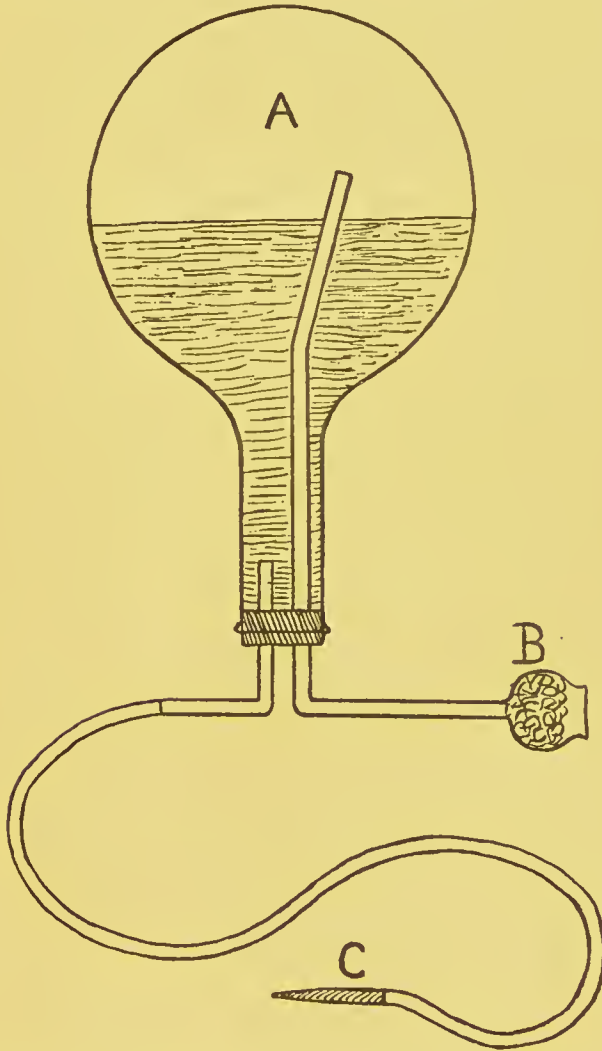
5. The lens is then forced out by gentle pressure on the lower edge of the cornea.

6. Any little pieces of cortex left in the eye are got rid of by gently rubbing the lower lid with a circular motion over the cornea, after giving a few minutes for the aqueous to collect ; or, better still, by irrigation of the anterior chamber—*vide infra*.

7. Small pieces of blood clot which may adhere to the wound are removed with forceps, the cut edges of the iris are replaced in the anterior chamber with a small spatula if they are caught in the wound, and the eye is douched with a warm antiseptic lotion, then bandaged. It should not be opened for two or three days, unless there is much pain or discharge, but the bandage may be removed daily, and the lids lightly sponged with an antiseptic lotion. The patient should remain in bed for several days, and live on slops to avoid chewing.

Cataract under ordinary circumstances is best operated on when ripe, but in many cases, as where a patient has to work for his living, and must go into the workhouse if his sight fails, it is right to run a slight additional risk in order to operate early. Two methods of dealing with

unripe cataracts are in common use—Forster's artificial ripening and M'Keown's irrigation method. I have no experience of the former, but have the fullest confidence in the latter. Forster's plan is to do an iridectomy (which



saves one step in the subsequent operation for removal of the cataract) and then gently massage the lens through the cornea. M'Keown's operation depends on the fact

that the difficulty in removing unripe cataracts is owing to their sticky nature, the lens not shelling out of the capsule easily, but pieces adhering. To get over this difficulty M'Keown injects water into the unripe lens with a hypodermic needle, to break up its tissue, then removes it in the ordinary way, and gets rid of the adherent masses of cortex by free irrigation of the anterior chamber. The objection urged by many surgeons against this procedure was that no adequate provision was made for antisepsis ; but I have devised a very simple piece of apparatus by which perfect asepsis is secured, and by which also the strength of the stream of fluid can be regulated exactly. My apparatus has been adopted by Dr. M'Keown, who has now employed it constantly for over four years with most satisfactory results. It consists of (A) an ordinary glass flask, holding about a pint. This is half-filled with a 2% solution of sodium chloride, which has been shown to irritate the tissues less than pure water. Through an india-rubber cork pass (B) a thistle-headed funnel, the head of which is filled with absorbent cotton wool, and (C) a short glass tube, connected with an india-rubber tube about eighteen inches long, ending in a hypodermic needle for the first procedure, or a silver nozzle for the second. The rubber tube and nozzle are sterilized, fastened on, and the tube clamped. The flask is then placed upright over a lamp and boiled, steam escaping through B, the contents being thus sterilized. After this all air coming into the flask through B is filtered by the wool, and the saline solution remains entirely aseptic. For use the flask is allowed to cool to about blood heat, easily recognised by the hand, and inverted, as in figure. On the clamp being removed, water flows from the nozzle with a forcedependent on the height at which the flask is held. When the needle is on, the water can be forced more quickly through it by

running the fingers down the tube—in fact, by a “milking” action. After removal of the lens, remaining cortex can be washed out by a gentle stream from the nozzle, inserted in the wound, with the greatest ease and safety. Many surgeons now omit the iridectomy when removing cataract, the advantage being that it simplifies the operation, and gives a better pupil; but in England the consensus of opinion is still in favour of doing iridectomy as the safer method.

(2) Congenital Cataract is not often discovered till youth, and though spoken of as congenital, may in some cases have formed in infancy. Such cataracts may be associated with other defects of the eyes, or may occur in otherwise normal eyes, and may be single or double. They are not generally progressive. They may be nuclear, but the most usual form is Lamellar (Zonular or Perinuclear) Cataract. This is due to defective development of one or more layers of the lens—these layers lying between the nucleus and cortex. Imagine an onion with all its layers transparent except one or two deep coats. On looking at it we should see a disc:—the circumference clear, the centre slightly cloudy, and between them an opaque ring where we see the milky layers edgeways. This is exactly what occurs in Lamellar Cataract. The defective development is generally due to rickets, associated with convulsions (Arlt). It is always bilateral (Fuchs).

*Treatment*—If vision is improved when the iris is dilated by atropine, it will also be improved by iridectomy. If not, the lens may be needled—that is, the anterior capsule torn with a needle passed through the edge of the cornea. The lens then breaks up, and is slowly dissolved in the aqueous. This operation is known as discission. This is only suitable for children under twelve or fifteen years of age. After needling, secondary glaucoma sometimes occurs

from raised tension caused by the swelling of the lens. If it occurs, a small peripheral incision is made in the cornea, and the aqueous, with some of the broken up lens matter, is allowed to escape. Iritis also may occur from irritation of the iris by the fragments of lens. It is treated with atropine. Both these complications are best guarded against by making only a very small tear in the capsule with the needle, specially the first time of doing. On repeating the operation, as must generally be done several times at intervals of a month or so, a larger tear can be made if the eye has been found tolerant.

(3) **Anterior and Posterior Polar or Pyramidal Cataract** consists in a small opaque patch at the centre of the anterior or posterior capsule of the lens. The latter is the commoner. Both may occur in one eye.

*Cause*—The Anterior may arise from contact of the capsule with a perforated ulcer of the cornea, when it does not interfere very seriously with sight. Either may form in deep-seated chronic inflammation of the eye, in which case vision is likely to be bad. They may be congenital. Neither form is likely to progress much, but sometimes the latter goes on to complete cataract, in which case operation does not offer much chance of success. When the former occurs alone, treatment is not generally required.

(4) **Traumatic Cataract** arises from wounds which perforate the anterior capsule, or blows which rupture it without injury to the cornea. If the eye is not otherwise injured, the lens may be slowly absorbed, as in discission. The younger the patient, the more likely is this to occur. Atropine drops should be used if the tension of the eyeball is not raised. If glaucoma comes on the lens should be removed, as in Senile Cataract.

**Dislocation of the Lens** may be congenital, in which

case it is often associated with other defects of the eye ; or it may be caused by chronic inflammation affecting the natural support of the lens—the Zonule of Zinn ; or, most commonly, by a blow on the eye. Certain signs point to this accident. The iris, when it loses the support of the lens, become tremulous. If only partial, the edge of the lens may be seen as a dark curved line against the red fundus when viewed with the ophthalmoscope. If complete, the lens disappears from sight, and its absence (aphakia) necessitates a strong convex glass to restore clear vision. The consequences of dislocation are serious, Glaucoma and Irido-Cyclitis often following.

*Treatment*—If possible the lens should be removed. Where it is out of reach, glasses are given to correct vision, and symptoms treated as they occur.

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## CHAPTER VII.

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### THE VITREOUS, RETINA, AND CHOROID.

#### THE VITREOUS.

Opacities in the Vitreous may be large or small, causing floating spots (muscae volitantes) before the sight, and more or less interference with vision. They are the result of deep-seated inflammation, or of hæmorrhages into the vitreous. They can be seen with the ophthalmoscope as black bodies against the red background of the fundus, and are distinguished from opacities in the lens by their irregular shape, and by their moving for a second or two when the eye is brought to rest after a quick movement.

**Liquefaction of the Vitreous** results from disease of the surrounding parts, and is generally associated with floating opacities, and recognised by their free movements. It is a serious sign as regards vision, for it indicates advanced deep-seated disease.

**Purulent Inflammation of the Vitreous** commonly follows perforating wounds. If the wound be large, or a foreign body be lodged in it, violent inflammation affecting the whole eye usually follows (panophthalmitis). Small punctured wounds may be followed by a less acute inflammation, in which the vitreous becomes infiltrated with pus cells, and the globe slowly softens and shrinks (phthisis bulbi).

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## THE RETINA.

**Inflammation of the Retina (Retinitis)** is characterised by more or less disturbance of vision, and by cloudiness of that membrane when viewed with the ophthalmoscope. There may also be hæmorrhage or exudation into its substance or from its surface. If the latter, the vitreous may become clouded and full of opacities. When a case is first seen in this stage it may be difficult to diagnose from opacity of the vitreous due to chronic cyclitis. In the latter, however, the opacities are more numerous and dense at the peripheral parts of the retina, while in Retinitis they are less so.

**Causes**—Retinitis is almost always associated with general disease, and as our knowledge increases the cases which we class as idiopathic become fewer and fewer. The more frequent causes are indicated below, under the varieties with which they are associated.

*Course*—Retinitis may occasionally run a quick course when slight, but, as a rule, it lasts for months or years.

*Consequence*—The common consequence of Retinitis is atrophy of the retina and the underlying choroid. This is not general, but occurs in irregular patches, which show as brilliant white, surrounded often by patches of pigment, caused by the migration of pigment cells from the patch undergoing atrophy, and their piling up at its sides. Atrophy of the retina destroys all sight where it occurs, so that the degree of blindness is directly proportional to the extent of the atrophy. *+ its proximity to Macula Lutea*

*Varieties* :—

ALBUMINURIC RETINITIS presents a most typical appearance, from which the presence of Bright's disease is often recognised. The changes in the retina are seen chiefly round the optic disc, which becomes swollen; the vessels are full and tortuous, the retina cloudy, and often hæmorrhages are present, but the typical appearance consists in a belt of small brilliant white spots of exudation surrounding the disc, at a little distance from it. The degree of blindness varies greatly, as it depends more on the uraemia generally present than on the retinitis. This form of retinitis may occur in any form of Bright's disease accompanied by albuminuria, but is most frequent in small granular kidney. It occurs only at an advanced stage of the kidney disease, and renders the prognosis very bad, most cases dying within six months or so, though some may live for years. The pathology is quite unknown. We may say that the retinitis is probably due to poisoning of the retina by uraemia, but this does not throw much light on the connection between the two. Both eyes are almost always affected, though not simultaneously.

SYPHILITIC RETINITIS occurs both in acquired and inherited syphilis, and is often accompanied by Iritis. It

presents no characteristic features, except that it is amenable to specific treatment. The prognosis is good as regards recovery from an attack, but it is likely to recur again and again, and eventually injure sight.—*Vide* Chap. XI.

HÆMORRHAGIC RETINITIS is characterised by the presence of numerous hæmorrhages into the substance of the retina, as well as the usual signs of retinitis—cloudiness of the retina, fulness of the vessels, and swelling of the disc. It is oftenest seen in cardiac disease, and is probably due to a secondary disease of the retinal vessels. Some cases end in Glaucoma. The disturbance of vision depends largely on the position and extent of the hæmorrhages. If there are several large ones round the macula lutea, there may be almost complete blindness. The prognosis is bad, as it generally runs on to atrophy of the retina, or glaucoma.

IDIOPATHIC RETINITIS, as remarked above, is becoming rarer as we know more of retinal affections, but some cases must still be put under this head where no general cause can be found, and a few cases occur in which a local cause, such as injury from a bright light, is recognised.

*Treatment of Retinitis*—Where a general affection exists, treatment must first be directed against it: such treatment is more properly described in text-books of medicine, and need not be entered upon here. The local treatment is largely symptomatic. The eyes must be protected by dark glasses, or by confining the patient in a dark room, and by abstention from all work. The use of leeches on the temple, or, better still, of wet cupping—or in feeble or delicate patients, dry cupping—is indicated when the retinal vessels are full and tortuous. Mild saline purgatives should be used frequently, and mercury and potassium iodide, even when no suspicion of syphilis exists.

Retinitis Pigmentosa is the very inappropriate name attached to a slow degeneration of the retina which is not

inflammatory in character, and which is not invariably associated with a deposit of pigment. The disease comes on in childhood, and progresses slowly and steadily for many years. The first symptoms of it are inability to see in a dim light, and a contraction of the field of vision, so that while central vision may remain good enough for a patient to read, peripheral vision may be so diminished that he cannot find his way alone. In most cases a curious deposit of pigment in the retina occurs sooner or later. This deposit is dense black, and scattered like fine moss all over the retina, but thickest at the periphery. Treatment is of no avail, and cases generally go on to total, or nearly total, blindness. Patients suffering from this disease are sometimes deaf mutes or idiots, or other members of their families may be so.

**Hæmorrhages** into the retina, apart from inflammation, occur under various conditions. In old people they point to atheromatous vessels, and are of serious import, as they are so frequently followed by cerebral hæmorrhage. They may occur in any altered state of the blood—anæmia, scurvy, &c. Large hæmorrhages into the vitreous humour, occur in otherwise healthy young adults without obvious cause. In girls they may be connected with disorders of menstruation.

*Prognosis*—Hæmorrhages are absorbed very slowly, generally taking months to disappear. They may leave behind them no sign, or the retina may be slightly altered in colour where they have been, or may have lost its sensibility.

*Treatment* is chiefly constitutional, attending to any condition present which may be held to account for the hæmorrhage. If the case be recent, wet cupping on the temple should be tried, or in old or feeble people, dry cupping. In a case recently under my care, a young lady

in whom very severe headache with hæmorrhage into the vitreous accompanied the menstrual periods, I found anti-pyrin, given at the onset of the headache, checked it at once, and there has been no return of the hæmorrhage.

**Embolism** of an artery in the retina is occasionally seen as a complication in affections which give rise to clots in the circulation—specially heart disease. It is characterised by sudden blindness of the whole or a part of the retina, according as the clot lodges in the central artery or a branch. The affected part of the retina becomes quite pale and loses its transparency, and, though it may regain this, the loss of sight is permanent. Multiple embolism of the retina is sometimes seen in pyaemia. Such cases are always fatal; but, if the patient lives long enough panophthalmitis occurs as a result of the septic embolism.

*Treatment*—In very recent cases of non-septic embolism, the cornea should be punctured with a needle, and the aqueous allowed to escape, in the hope that the lowering of tension, and consequent increased force of circulation, may drive the embolus into a smaller branch of the artery, where its effect will be less hurtful.

**Thrombosis** of a retinal vein may occur, either in the central vein or a branch. It is seen oftenest in cardiac cases, but may occur as a complication in orbital disease. The retinal veins are seen to be greatly distended, the arteries nearly obliterated, and hæmorrhages frequent. Like Embolism, it totally destroys vision in the affected part of the retina.

**Detachment of the Retina** is by no means a rare condition. It usually comes on suddenly. The first complaint of the patient is that when looking with one eye objects are dim and distorted. As the great majority of detachments begin at the upper part of the eye, the lower part of the field of vision is first affected, but the normal course

is an increase in the detachment, till eventually the retina becomes like a convolvulus flower, having its stem at the optic disc, and its edge attached to the globe at the ora serrata. On ophthalmoscopic examination some part of the retina is seen to lie farther forward than the surrounding parts, showing an irregular and wavy surface of a bluish or dirty grey colour. The vessels seem darker than normal, and need a constant change of focus to follow them. When the detachment is very extensive, the retina may be seen without the ophthalmoscope, through the lens, and may be illuminated by focal illumination. When the detachment has lasted some time, atrophic changes generally take place, and the field of vision becomes contracted. When occurring, as it usually does, above, the detachment almost always spreads downwards in time.

Cause—Detachments come under four headings—1st. Those complicating high myopia. In Germany these form 60% of the whole, but not so many in this country. 2nd. Traumatic, generally complicated by hæmorrhage, &c. 3rd. Idiopathic, without known cause. In these both eyes are often affected, though, perhaps, months or years apart. 4th. Those due to tumours. The last class is quite distinct from all the others, as the detachment may occur at any part of the retina, is at first small and circumscribed, and not loose and wavy. The tension of the eyeball is a most important point: for, while it is almost always lowered in ordinary detachments, it is raised when an intraocular tumour occurs. (*Vide* Sarcoma of Choroid.) Detachments are more common in the old than in the young, and in men than in women.

Pathology—The whole thickness of the retina is not detached, as the hexagonal pigment layer remains attached to the choroid. The pathology of detachment is not thoroughly understood, but it seems as if it were often

due—in the 1st and 3rd classes—to shrinking of the vitreous, and preceded by a rupture in the retina. At any rate, the proportion of cases in which a rupture is found if carefully looked for is very considerable. The theory now in favour is that the first step is the shrinking of a diseased vitreous, with accumulation of fluid in spaces between it and the retina. The shrinking vitreous drags on the retina, which eventually ruptures, and the fluid rushing in behind causes its sudden detachment. There are, however, still many points to be cleared up.

*Treatment* is most unsatisfactory. The routine treatment in recent cases is to keep the patient in bed for some weeks, on his back, and use pilocarpine subcutaneously. Some few cases may improve, but even these are pretty certain to relapse. Various operative measures have been proposed—all alike to fail. The latest method is to perforate the sclera behind the detachment with a flat needle, insulated to within a few millimetres of its point, and connected with the positive pole of a battery, the negative being applied to the neck, and a current of two or three milliamperes passed. It remains to be seen whether this will meet with greater success than former methods.

Glioma of the Retina is the only neoplasm of the retina, and is by no means common. It is a disease of early childhood, and is sometimes congenital. Owing to the age of the patients attention is not usually called to it till the growth has advanced so far into the vitreous as to be seen as a white or yellowish mass through the lens. At this time blindness of the eye is complete, and the tension of the eyeball very high. The stony hardness of the globe is an important point in distinguishing it from suppurative choroiditis, sometimes called Pseudo-Glioma.

*Pathology*—Glioma is a soft white tumour, composed mostly of round cells. Histologically it bears a strong

resemblance to round-celled sarcoma, but clinically it is quite distinct. It begins as disseminated patches in the granular layers of the retina, grows rapidly, involves the choroid and optic nerve, and, after filling the eyeball, bursts out.

*Treatment*—As soon as glioma is diagnosed, the eyeball should be removed, dividing the optic nerve as high up as possible. Early removal gives good hope of life; but if not undertaken till a later stage recurrence is probable. If the disease is allowed to run its course, death occurs from extension to the brain. Metastasis is uncommon. Both eyes are often attacked, either simultaneously or one after the other.

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## DISEASES OF THE CHOROID.

Choroiditis—Inflammation of the Choroid is a common and serious affection, occurring at all ages. It is well to remember that this membrane, where visible at all, is only seen through the retina, consequently the earliest stages of diseases in it are hidden from observation. As with other forms of inflammation, authors have constantly exercised their ingenuity in inventing new sub-divisions of choroiditis, apparently believing that the man who shows two varieties of inflammation to exist where one existed before is as great a benefactor to his race as the man who causes two blades of grass to grow where only one existed previously. With the exception of purulent choroiditis, however, no very distinct and clear-cut varieties exist, though several clinical types may be described. Leaving aside Purulent Choroiditis, the appearances have much in common in all types. The most marked character in the appearance of Choroiditis is its patchy look. It occurs always in

patches, larger or smaller, with areas of normal looking membrane between them. These begin as patches of effusion, which are slowly absorbed, leaving atrophied areas, where the shining white sclerotic is seen. Round each of these areas the retinal pigment is generally heaped up in little black masses, over which the retinal vessels are seen to run. Often patches may be seen side by side in different stages—some new, others old and atrophied. The degree of interference with vision depends on the extent of the patches and their proximity to the macula lutea.

*Cause*—Roughly speaking, about half the cases of Choroiditis are due to syphilis, inherited or acquired. In the former a type known as disseminated choroiditis occurs either in intrauterine or very early life. Small atrophied patches are seen over the whole fundus, but no recent patches of exudation. In adults all stages are often seen at once. The patches may be disseminated, or may be confined to one region. In old people they are sometimes found grouped round the macula, destroying all direct vision, though leaving the general field good enough for the patient to make his way about alone. Some cases are associated with scleritis either at the anterior or posterior part of the globe. When anterior, there is generally sclerotising keratitis. When posterior, it is usually associated with high myopia, and as the whole coats of the eye are inflamed, they become weakened and bulge out, forming a posterior staphyloma, and thus increasing the myopia. In cases where no syphilitic history can be traced, and there is no history of inflammation spreading from neighbouring parts, no cause may be found.

*Treatment*—When a case of Choroiditis is seen, the first thing to do is to dilate the pupil and search the fundus well. If nothing is seen but old patches of atrophy, with

no signs of recent disturbance, treatment is quite useless. If, however, any patches of exudation are seen, or any signs of recent inflammation, active treatment is called for. Mercury and Iodide of Potash should be given in all cases, even where no specific history is to be found. In the latter event, however, they need not be so long maintained, but Iodide of Iron may be substituted. Dry cupping on the temple, smoked glasses to protect the eyes from glare, and avoidance of all use of the eyes for near vision should be ordered, and attention paid to the general health.

*Prognosis*—Both eyes are usually affected. In old cases, where no recent inflammation exists, the case is likely to remain in the same condition. If any recent signs exist there may be a recrudescence of active inflammation at any time, and the patient should be kept under observation. In active cases, even when severe, patients may be comforted by the fact that absolute blindness is not likely to result—though all working vision may disappear.

**Purulent Choroiditis** may be caused by operation wounds, or the penetration of foreign bodies into the eye; or may arise as a complication in cerebro-spinal meningitis in children. Purulent inflammation may extend to the choroid from the anterior structures of the globe. When arising in the choroid it may remain confined to it, taking the form of a slow inflammation, leading to gradual softening and shrinking of the globe (phthisis bulbi). The yellowish-white reflex from the purulent exudation in the vitreous has sometimes been mistaken for Glioma of the Retina, causing the name of Pseudo-Glioma to be given it. The low tension of the globe is the great point of distinction, however, and no real difficulty should exist in the diagnosis. Purulent Choroiditis may also be acute, in which case it is likely to spread to other parts of the eye—panophthalmitis resulting.

*Treatment*—In the slow form nothing can be done to stay the course of the affection, which goes on to complete loss of sight, except in cerebro-spinal meningitis, when a few cases may recover useful vision. I lately saw one such in Professor Siemisch's clinic at Bonn. In Panophthalmitis treatment consists in allaying pain by hot stuping, or the administration of morphia if necessary. Enucleation should not be done till active inflammation has subsided. The risk of sympathetic ophthalmia is slight, or nil, when once general acute inflammation has set in.

**Coloboma** of the Choroid is a congenital abnormality liable to be mistaken for disease. It consists of an absence of the choroid over a narrow sector of the fundus, the centre being at the optic disc. It is often associated with coloboma of the iris, or other defects of development.

**Sarcoma of the Choroid** is always primary, and is by far the commonest neoplasm in the eye. It may occur at any age, but is commonest in later life. It begins as a small nodule in the choroid, which interferes little with sight unless it is close to the macula. As it grows it pushes the retina in front of it, causing a detachment and increase of tension. In some cases this increase is so rapid that the symptoms of acute glaucoma are produced. After a time it perforates the sclera, generally at the corneo-scleral margin, and grows rapidly outside the eye, filling the orbit. The bones are rarely affected, but it may extend up the optic nerve. Pain is greatest before perforation, when the tension is high, and again when the growth in the orbit becomes so large as to push the eye forwards, causing Proptosis. By this time secondary growths have generally appeared, most commonly in the liver, and a fatal result follows. The whole course is never over five years, and is generally much less. It is very seldom bilateral.

*Pathology*—Choroidal sarcoma is round or spindle celled, and may be pigmented or not, according to its point of origin, as pigment, if present in the tumour, is derived from proliferation of that physiologically present. The great majority of cases arise near the optic disc, and are pigmented or melanotic. Non-pigmented, or leucosarcomata, are found anteriorly ; they are always round-celled, and most malignant.

*Diagnosis*—Attention is not often called to the eye at an early stage, but if it is, the sarcoma may sometimes be recognised as a small dark nodule near the optic disc, causing a little local detachment of the retina and raised tension. Later, when the detachment is greater, it is distinguished from simple detachment by the pain, absence of tremulousness, and less sudden onset, but specially by the raised tension. From glioma it is distinguished by the fact that the former occurs in the first decade of life, and is always non-pigmented. The lymph glands near the eye are affected in glioma, but not in sarcoma. From acute glaucoma it is distinguished by the fact that pain and loss of sight come on suddenly together in glaucoma, while there is generally some loss of sight before there is any pain in sarcoma. In glaucoma the other eye is nearly sure to be more or less affected, and there will have been premonitory symptoms.

*Treatment*—The only treatment of any avail is enucleation. If this is done before the tumour has perforated, the chance of local recurrence is not great—only about 4%. But the great danger is from metastasis, which proves fatal in the great majority of cases. According to Professor Fuchs, it is just as likely to occur after early enucleation as after late. If the growth is advanced, the whole contents of the orbit should be removed.

**Carcinoma of the Choroid** is exceedingly rare, and is always metastatic. Early enucleation is the only treatment.

## CHAPTER VIII.

## THE OPTIC NERVE.

Inflammation of the Optic Nerve occurs in two very different forms: the first appearing at the peripheral termination of the nerve, when it is known as Optic Neuritis, and the second in its course between the brain and the eye, shown only by its results—Retro-bulbar Neuritis. The latter is always chronic.

Optic Neuritis (Papillitis, Choked Disc) is only recognised by the ophthalmoscope. It is more often discovered by the physician than by the ophthalmic surgeon, on account of its mode of occurrence (*vide infra*) and the frequency with which it exists without interfering with vision. The appearances vary according to its intensity, but the chief points are—1st, a wooly appearance of the disc, the sharp, clear margin being lost; 2nd, swelling and hyperaemia of the disc, even to the extent of projecting in a mushroom shape from the level of the fundus; 3rd, fulness and tortuosity of the veins, in comparison with which the arteries appear insignificant. The vision is not usually affected at first, and in slight cases may not be so at all, while in other cases it may be reduced even to total blindness. It is more likely, of course, to be seriously affected where the retina as well as the disc is inflamed (neuro-retinitis). “Choked disc” is sometimes recognised as a minor form of optic neuritis.

Cause—The commonest cause is tumour of some sort in the brain, in which case the papillitis is practically always double. Tumour in the orbit or near the course of one nerve may produce unilateral papillitis. Meningitis,

specially tubercular, is the next commonest cause. Abscess in the brain, lead poisoning, and acute febrile diseases are sometimes accompanied or followed by papillitis. Neuro-retinitis is a frequent complication in Bright's disease (*vide* Retinitis). Syphilis may cause papillitis, usually by a gumma or other growth in the brain. Menstrual disturbances, and specially suppression of menstruation, are accountable for some cases ; and after all these causes are exhausted, some cases remain which must be classed as idiopathic—from cold, &c.

*Pathology*—Besides the congestion and oedema of the papilla, there is infiltration of lymph cells, proliferation of nuclei, and degeneration of nerve tissue. The mode of occurrence of these changes has given rise to much discussion, for the lesion causing them may be very small and in a distant part of the brain. One explanation is that when a cerebral tumour is present, there is an increase of the intracranial fluid, and consequent raising of intracranial pressure. The fluid fills the sheath of the nerve (which is a continuation of the meninges), and distends it, causing pressure and, consequently, swelling and inflammation at its distal end. Another explanation is that there is a neuritis, causing only microscopic changes, which spreads down the nerve. A really satisfactory explanation is not yet forthcoming, as all hitherto advanced involve difficulties. Those cases unconnected with organic disease must be looked on as instances of idiopathic neuritis. According to Dr. Gowers, some of these seem to be due to functional brain disease combined with hypermetropia, though the reason of the connection is quite unknown.

*Prognosis*—This is decidedly bad in cases arising from organic disease, as the papillitis, after lasting for some time, usually passes into Optic Atrophy (*vide* infra). When it arises from meningitis, specially in children, the prog-

nosis is better, many cases retaining useful vision. In cases which arise from other causes the prognosis, though varying with the severity, is on the whole decidedly better than where cerebral disease exists.

*Treatment* must be directed first to any constitutional cause which may be present. Mercury and Iodide of Potash should be tried in all cases. As regards direct treatment, little can be done. Wet or dry cupping on the temple and subcutaneous injection of Pilocarpine may be tried, and much use of the eyes avoided. Tonics should be given as the neuritis subsides.

**Retro-bulbar Neuritis** (Central Amblyopia) is a chronic inflammation of the optic nerve in its course in the orbit, characterised by a loss of central vision (central scotoma). The scotoma is generally *negative*, that is, it appears to the patient simply as an absence of visual sensation in a certain direction, and not as a black spot or area, which would be a *positive* scotoma. This rare form of neuritis is found both in men and women, and, as a rule, affects one eye only. It is often confused with toxic amblyopia, but the latter affects both eyes, is more amenable to treatment, and in it a history is obtained pointing to the toxic character. The scotoma also differs in character, having a great tendency to extend towards the nasal side, causing a loss in the field of vision on the temporal side. Exposure to cold is supposed to cause this affection. Treatment may be carried out on the same lines as that of papillitis, but the results are unsatisfactory, and the prognosis bad as regards vision.

**Optic Atrophy** is characterised by a slow wasting of the nerve substance and capillaries seen at the papilla, and a progressive loss of vision, ultimately complete, beginning at the periphery of the field of vision. Colour vision is early affected. The disc loses its pink colour and becomes

white or grey, and the normal physiological cupping becomes deeper. The vessels may be reduced in size, but are not always so. Atrophy following inflammation of the papilla is known as "consecutive" atrophy (other cases being called "simple" atrophy). The vessels are always reduced in size in the former. Simple atrophy may be primary or secondary. When primary, the loss of vision and the changed appearances of the papilla proceed together; when secondary, vision is lost before changes are seen. Primary atrophy is often hereditary, is almost always bilateral, and 75% of cases are seen in men. Probably about 50% are due to Tabes Dorsalis, of which it is a most important sign. It may be the first sign of the general disease, and if optic atrophy is found associated with lightning pains, there is little room for doubt in the diagnosis. The disc in these cases is generally grey in colour, with no diminution in the size of the vessels. Other spinal affections may also cause atrophy; and syphilis, acute specific fevers, menstrual disturbances, and many other causes have been indicated more or less frequently. Secondary atrophy follows lesions of the optical centres or fibres. Pressure on the nerve during its course will cause it, and embolism and thrombosis of the central artery. Blows on the head may bring on a slow atrophy—as in the case so vividly described by Rudyard Kipling in "The Light that Failed."

*Pathology*—The nerve may be found much reduced in thickness, the nerve fibres being absent, and their place filled by fat granules and other evidences of degeneration; or it may be of normal size, but composed of connective tissue in place of the nerve fibres. The retina does not share in the atrophy, except in its inner layers—the nerve fibre and ganglion layers.

*Diagnosis*—Mere paleness of the papilla is not sufficient

to found a diagnosis upon, unless accompanied by both lessening of the acuity of vision and concentric contraction of the field of vision. Colour vision should also be tested.

*Treatment*—If constitutional disease is present, treatment must be directed first against it. The only useful treatment for the atrophy is hypodermic administration of strychnine, beginning with  $\frac{1}{40}$ , increasing slowly to  $\frac{1}{12}$  gr. daily. If benefit does not result in ten days or a fortnight, it may be given up.

## CHAPTER IX.

### AMBLYOPIA AND AMAUROSIS.

Cases frequently occur in which vision is interfered with without obvious pathological changes in the eye. If vision is merely lowered, these cases are classed as Amblyopia ; if lost, as Amaurosis ; but the terms are often used rather loosely. The sub-divisions of these form a very heterogeneous group.

Toxic Amblyopia is one of the commonest forms. It is characterised by a loss of central vision in both eyes, and of colour vision, which loss is due to some toxic agent. Sight is generally regained when the cause is removed. Much the commonest cause is excessive use of tobacco, combined with frequent doses of spirits. It undoubtedly occurs sometimes, though rarely, in heavy smokers who abstain from alcohol, and still more rarely in non-smokers who are constant spirit drinkers. The common strong tobacco smoked by working men is said to be more liable to cause it than lighter sorts, but this is probably due to

the fact that working men so often smoke on an empty stomach early in the morning. The subjects of toxic amblyopia are, as a rule, men over forty years of age, who have smoked heavily, and constantly indulged in spirits, for many years. The first symptom noticed is often an inability to distinguish colours—for instance, a man will say he has found himself confusing shillings and sovereigns. When more advanced a negative scotoma (see Retro-bulbar Neuritis) develops; it is small, oval, involves central vision, and is exactly the same in both eyes.

*The Pathology* is unknown. It has often been classed with Retro-bulbar Neuritis, but the exact similarity of the scotomata in both eyes in toxic amblyopia is strong evidence against this view, and in favour of the view that it is due to poisoning of some of the brain centres. The prognosis, too, is much better than in Retro-bulbar Neuritis.

*Treatment* is satisfactory if the case is not of very long standing, and if the patient has strength of mind to carry it out. But total abstinence from tobacco and alcohol is the first and most important point, and, needless to say, there are men who prefer anything short of total blindness to such a privation. Strychnine should be given hypodermically in the temple, beginning with about  $\frac{1}{40}$  grain daily, and continued for several weeks, gradually increasing the dose to  $\frac{1}{12}$  grain, unless the physiological effects of the drug appear.

Simulated Amaurosis is sometimes seen, most frequently in soldiers or police who want to avoid duty, and in young women who wish to attract a little attention and gain the sympathy of their friends, or possibly to give some trouble. It is seldom they attempt such skilful acting as is required to feign total blindness of both eyes, and when only one is complained of a good deal of acuteness may be necessary

to prove the fraud, even when suspected. Several tests should be tried in each case, for it is decidedly awkward, for instance, for a medical man to certify a man as unfit for duty, on account of blindness, whose sight may afterwards be proved perfectly normal.

*The Diplopia Test* consists in holding a prism in front of one eye, the patient looking at a light some distance off. If he sees two images, he is using both eyes ; but the patient may be too sharp, and by a casual wink of the supposed bad eye discover what he *ought* to see, with one eye only, and then declare he sees that. A modification of this plan is to cover the supposed bad eye, and hold a prism base down in front of the other, so that the edge of the prism crosses the middle of the pupil. Two images will now be seen with the one eye, then the hand is quickly removed from the covered eye, and the prism moved up, so as to cover the pupil of the other. If both eyes are in use, two images will still be seen. There are other more elaborate tests, in which different lenses or coloured glasses are placed in front of the eyes, but these are only suitable for ophthalmic hospitals, and the general practitioner who has a case to test can easily invent tests for the occasion. If answering is not *prompt*, it is very suspicious.

**Hysterical Amblyopia** takes many different forms ; not merely lowering or loss of vision, but interference with vision by subjective sensations of all sorts. It is only by a careful consideration of the general conditions that Hysterical Amblyopia can be detected. Its treatment is, of course, that of hysteria generally.

**Reflex Amblyopia** may arise from irritation in any part of the body, but oftenest from the teeth. It may take the form of subjective sensations of light or colour, or hyperaesthesia of the retina.

**Congenital Amblyopia** is not infrequent. The vision in both eyes may be sub-normal, but is generally worse in one than the other. Where the vision in one eye is much less than the other, it often comes to squint eventually ; and, on the other hand, in old cases of squint, vision is much lowered in the squinting eye. The vision in astigmatic eyes is often sub-normal, even when the astigmatism is fully corrected by glasses. A very useful test for an *old* amblyopic eye has lately been pointed out. If the patient is told to close first one eye and then the other, it will be found that he can close the *bad* eye alone, but not the good eye : the reason being that the power of closing one eye is only attained by practice, and as the patient has never desired to use his worse eye alone, he has never practised closing the good one.

**Amblyopia and Amaurosis from Central Causes**—Some such cases come on suddenly during pregnancy or convalescence from acute febrile diseases, and are then probably due to uraemic or other poisoning of the centres. The prognosis in these cases is good. If following injury to the head, as sometimes happens, prognosis is very bad.

**Night Blindness** is a peculiar condition in which the retina loses its adaptability to varying intensities of lights, so that objects are seen only dimly, or not at all, in a weak light, which are easily seen in a strong. It is often the result of long dazzling of the eyes by a strong light, and is sometimes associated with some congenital affection, though why this should be so is unknown. The treatment is absolute rest to the eyes in a dark room for several days, then a slow increase in the light.

**Hemianopsia** is a loss of half the field of vision, generally in both eyes, due to central causes. It is most often homonymous—that is, the right or left half of the field is lost in each eye, so that one side of the field of vision is

completely lost. In temporal hemianopsia, less common, the temporal half is lost in each eye, so that the right eye sees only the right half of the field, and the left eye the left half. Nasal hemianopsia occurs seldom or never. The localisation of the lesion is often possible, but the subject is too complicated to enter upon here. The cause of the lesion is generally a cerebral hæmorrhage, and the prognosis bad.

**Colour Blindness**—About three or four men in every hundred, and one or two women in every thousand, have defective colour vision congenitally, while it is also found as a symptom in toxic amblyopia and in optic atrophy. The theories of colour vision are very varied, and as far as the recognition of the existence of colour blindness goes, are not of much practical importance. It is, however, important that every medical man should be able to apply tests for colour blindness, for cases often occur, specially among railway employees and sailors, in which a certificate of sound colour vision is necessary. The most certain and simple test is Holmgren's, or some modification of it, such as that to be found inside the back cover of this book. This consists of a number of small skeins of thread, of all colours, arranged in complete confusion—the first skein in each of the first three rows being a test skein. The first test skein is a light pure green, the second a pale pink, and the third a strong pure red. The first is the most important. The person to be tested is shown this, and asked to pick out other skeins to match it, without naming the colours. If he does this correctly, he has normal colour vision. If he picks out other colours, such as pale yellow, brown, or grey, his colour vision is defective. The other test skeins may be given as confirmatory, if he fails to match No. 1 correctly. If the defect is great, he will put wrong colours with these also; but if only slight, he may succeed with

these, though he has failed with the first. A person who fails with the first, even though he succeeds with the others, should never be passed for a responsible post such as engine driver, where confusion of signals may lead to disastrous results.

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## CHAPTER X.

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### ERRORS OF REFRACTION.

For the proper understanding of this subject an elementary knowledge of Optics is necessary, and the reader who is at all doubtful of his knowledge will do well to refresh his memory by glancing over the chapters dealing with refraction and lenses in some text-book of Physics.

The character of the eye as an optical instrument depends on the varying shapes and densities of the structures through which light passes to reach the retina—the *refracting media*, as they are called. In practice we find that errors of refraction are practically always due to departures from the normal shape of these structures. The hardening of the lens with age which causes Presbyopia is a physiological process, so that its result can not be called an error of refraction, though it necessitates the wearing of glasses for near vision.

**The Dioptric System** is the system of numbering lenses now almost universally in use. The unit—one dioptre—is a lens whose focal distance is one metre (almost exactly forty inches). A lens of two dioptres, written 2 D, has a focal distance of half this—that is, 50 cm. or 20 ins. A

3 D lens has a focal length of 33·3 cm. or 13·3 ins., and so on. The focal length of any lens whose strength in dioptries is known, can be at once told in centimetres by dividing the number of dioptries into 100, or in inches by dividing the same into 40. For instance, a lens of 8 D has a focal length of  $\frac{100}{8}=12·5$  cm. or  $\frac{40}{8}=5$  ins. When the lens is convex, it is called a *plus* lens; when concave, a *minus* lens, so that  $-2$  D means a concave lens of 50 cm. focal length, and  $+2$  D a convex lens of the same focal length.

**Normal Vision** varies within wide limits, and the arbitrary standard chosen is a decidedly low one, The acuity of vision (written simply V) is said to be normal when plain black Roman capitals, on a white ground, well illuminated, can be read at such a distance that the height of each letter subtends an angle of 5' at the eye, and the breadth of the limbs an angle of 1'. In testing this the patient is placed at a distance of six metres or twenty feet from the test type. Cards are sold by all opticians, on which are letters varying from those which ought to be read at sixty metres (or two hundred feet), down to those for six metres (or twenty feet). Each eye is tested separately, the other being covered. If at six metres the patient reads the lowest line of type, marked  $D=6$  (that is, to be read with normal V at a distance of six metres), we say  $V=\frac{6}{6}$ , or, if we are counting in feet,  $V=\frac{20}{20}$ —that is, it is normal. Supposing the patient can only read the largest letter, marked  $D=60$ , we say  $V=\frac{6}{60}$ —that is, he can only read sixty metre type at six metres. If he was not able even to see this till he walked up to within two metres of the card, we should say  $V=\frac{2}{60}$ . Lower degrees of V we mark as "counts fingers at so many feet," or "sees hand at so many feet." A patient may have normal V for distance, and yet very defective for near work, so he must further

be tested by seeing *how near the eye* he can read very fine type. To be read at all, this must be seen clearly, so this gives us his *near point*, or point of greatest accommodation. This varies with the age of the patient, being normally as follows :—

DISTANCE OF NEAR POINT (after Landolt).

Age.	Inches.	Centimetres.	Greatest Possible Accommodation.
10	2·8	7·0	14·0 D.
15	3·3	8·3	12·0 D
20	4·0	10·0	10·0 D
25	4·6	11·7	8·5 D
30	5·6	14·0	7·0 D
35	7·0	18·0	5·5 D
40	8·7	22·0	4·5 D
45	11·0	28·5	3·5 D
50	16·0	40·0	2·5 D
60	39·5	100·0	1·0 D
70	157·0	400·0	·25 D

From this it will be seen that the near point recedes more and more rapidly as age increases, and the power of accommodation for near objects gets less and less. This is due to gradual loss of elasticity in the lens, and when it reaches such a degree that clear vision is no longer possible at ten or twelve inches, it is known as Presbyopia. Looking at the table we see that a person of forty-five can normally exercise 3·5 D of accommodation—that is, he can focus objects at 11 inches or 28·5 cm. clearly on the retina. But this is only possible when straining his accommodation to the utmost, and such strain cannot long be maintained, so that he cannot read comfortably

at less than fifteen or sixteen inches, where he is exercising only 2.5 D of accommodation. In practice we find that only about two-thirds or three-fourths of the total accommodation can be continuously exercised, and as about 3.5 D of accommodation is necessary for ordinary reading at ten or twelve inches, we must supplement the failing powers by a convex lens of strength sufficient to bring rays from a point ten or twelve inches off to a focus on the retina, without calling on more than a fair portion of the accommodation to be exercised. In the case of the man of forty-five, who can comfortably exercise 2.5 D of accommodation, convex lenses of 1 D will be sufficient, and will give him a near point of eleven inches for ordinary work. Supposing he is sixty years old, and his near point is about forty inches away, he can only exercise 1 D of accommodation, which is quite insufficient for reading; but by giving him lenses of +3.5 D, we bring him back to a strength of accommodation the same as he had twenty years ago, namely, 4.5 D, which enables him to read his newspaper with ease at ten or twelve inches. The treatment of Presbyopia, then, is to find the distance of the near point, and order convex glasses of sufficient strength to bring back the near point to eight or ten inches.

*General Examination for Errors of Refraction*—Supposing we are dealing, not with a case of Presbyopia, but with a person, young or old, whose sight is deficient, and in whose eyes no disease has been found to account for the defective sight, a certain routine should be followed in making our examination. The acuity of vision of each eye separately is first found by placing the patient at six metres from a test card, and finding what type he can read. Then different glasses are tried, and their effects noticed; and then the “near point” is found by seeing the nearest point at which small type can be clearly seen. In an

adult this may be sufficient in many cases to show us the character and amount of the defect of refraction, but in children and young people a further examination must always be made after the eyes have been under the influence of atropine for several days, to paralyse accommodation. The reason for this is that in young people the real nature of the error may be entirely masked by spasmodic contraction of the ciliary muscle, converting a case of hypermetropia into apparent myopia. The second examination should be similar to the first, for distant vision ; but near vision cannot be examined, of course, when the ciliary muscle is not acting. This examination by trial glasses must be checked by an objective method, not depending on the patient's statements ; and the best and easiest of such methods is the one variously known as the shadow test, Keratascopy, or Retinoscopy. The examiner sits in front of the patient, about four feet from him, and with a slightly concave mirror reflects a beam of lamplight into the dilated pupil. The mirror is then rotated slightly, first on a vertical axis, making the spot of light pass across the eye from side to side, and then on a horizontal axis, making it move up and down. In an ordinary normal or emmetropic eye, the spot of light is seen to move across the fundus of the eye in a direction *contrary* to that in which it moved on the face, and the same phenomenon is seen in hypermetropia, while in myopia the light in the eye moves in the same direction, as on the face. In the former case it moves, we say, *against* the mirror, in the latter *with* it. We then try what glasses are needed to alter the direction. In a normal eye the light (or shadow, as it is often less correctly put), moves against the mirror, but a convex glass of .5 D held in front of it converts it into a myopic eye, and the light moves *with* the mirror. If it moves with it in the

first instance, showing that the eye is myopic, a concave glass will be found which will neutralise the myopia, converting it into emmetropia, and so reverse the direction of the light. In some cases the light will be found to move in contrary directions, in two meridians at right angles to each other (*vide* infra, Mixed Astigmatism). When an examination has been made under atropine it is seldom advisable to prescribe glasses at once, but the results should be confirmed at a subsequent examination made a fortnight or so later, when the effects of the atropine have quite passed off. In many cases of young people of say twenty or twenty-five we may wish to make an objective examination with the accommodation paralysed, but not like to keep them from work for so long a time as is necessary with atropine. In these cases homatropine and cocaine drops (2% of each) may be substituted. Their effect is produced in half-an-hour or so, and passes off in a day, while it is quite sufficient in young people (*not* children) who have not been straining their sight very specially.

**Hypermetropia** is an error of refraction caused by a shortening of the eyeball in its antero-posterior axis, making it approximately orange shaped. Rays of light from a distance, which are brought to a focus on the retina in a normal eye at rest, in this case come to a focus behind the retina, so that the hypermetrope must exercise his accommodation to focus even distant objects on the retina. Supposing the eye is a millimetre too short, he would have to exercise about 3 D of accommodation for distant objects, and then in looking at an object half a metre away, where an emmetrope must exercise 2 D of accommodation, this hypermetrope would have to exercise  $2 + 3 = 5$  D of accommodation. The result is that his eyes are fatigued by near work, and a train of symptoms set in which are very characteristic.

*Symptoms*—The patient says he can see quite well at a distance, but his eyes are easily tired at near work. They feel all right in the morning, but gradually get “weak” as the day goes on, and are worse when working by artificial light. He may complain of constant evening headaches, pain in the eyes or lids, or up the side of the nose, all getting worse as the day goes on, till work is stopped, when they gradually lessen. A very frequent complaint is sudden blurring of the letters or work he may be looking at; it is caused by a sudden giving way of the overstrained ciliary muscle, just as the arm, if held straight out from the shoulder for a little time, will ultimately drop to the side, and though raised again, will again drop when the muscles become too fatigued to act. On testing the patient's near point with fine print, we find that it is further off than it should be for his age, and the difference between the actual and the normal corresponds with the hypermetropia present. For instance, if the patient is thirty years old, he should be able to exercise 7 D of accommodation, and see clearly at five and a half inches (see p. 75); but if he can only see clearly at eleven inches he is exercising only 3·5 D of accommodation, and is hypermetropic to the extent of 3·5 D. This method is of use in confirming other methods, but is not sufficiently exact for general use, as the table given on page 75 is only a general approximation, and not scientifically accurate. In examining such a case we may find distant vision normal, but this is obtained by exercise of accommodation, and remains as good, or improves, when convex glasses are used, which would not be the case with an emmetrope. To ascertain the amount of the error therefore, we find *the highest convex glasses* with which distant vision is good. This is called the *manifest hypermetropia*—there is a further amount of the total hypermetropia known as the

*latent* hypermetropia, and this is only discovered under atropine. In a child the latent may be the larger portion, but it decreases as age increases, and becomes unimportant in later life. In a child glasses should be given sufficient to correct the manifest and about half the latent hypermetropia. For instance, if on our first examination we find a child will take  $+2$  D glasses (convex glasses of two dioptries), and then under atropine will take  $+6$  D, we should order  $+4$  D. In an older person it is sufficient to fully correct the manifest hypermetropia. If with convex glasses the hypermetrope has not a visual acuity of  $\frac{6}{6}$ , we must test for astigmatism (*vide infra*). Hypermetropia is congenital, and does not increase, but it may not show itself for years, tonic spasm of the ciliary muscle being sufficient to neutralise it when the hypermetrope is young. In high degrees of hypermetropia *strabismus* is often developed, specially if one eye is weaker than the other. When glasses are ordered for children, they should be worn constantly, except when games are played in which they might be broken.

**Myopia** is an error of refraction caused by a lengthening of the eyeball in its antero-posterior axis, so that it becomes egg shaped. Parallel rays come to a focus before reaching the retina, and no effort on the part of the myope can alter this, so that distant vision is never clear without concave glasses. Some improvement in the sharpness of the image on the retina is obtained by lessening the aperture through which light enters the eye (equivalent to inserting a diaphragm in a photographic lens or microscope), so the myope often nearly closes his lids, or "screws up his eyes" in a very characteristic way. Near vision is not affected in moderate myopia, but in some cases the error may be so great that the myope can see distinctly nothing more than a few inches from his

eyes. Myopia is measured by the strength of the concave glass required to bring parallel rays to a focus on the retina. The farthest point at which vision is distinct we call the *far point*. Supposing this is one metre off, a concave glass of 1 D is required to make distant vision clear, and we say the patient has one dioptré of myopia, which would be very slight. If the far point is only 25 cm. off =  $\frac{1}{4}$  metre, -4 D will be required, and this is the measure of the myopia. The *near point* is approximated to the eye just as much as the far point, so that a myope of sixty years of age with 4 D of myopia would be able to see clearly at  $1+4=5$  D = 20 cm., while a normal sighted man of the same age could only see clearly at 1 metre. Myopia is largely a product of civilisation, and is found most where school work is hardest. Unlike hypermetropia it is rarely congenital, but comes on in childhood, and increases specially in youth. If not checked, it may, and often does, increase till it becomes not merely an error of refraction, but a disease of the eye, for the whole back of the eye suffers from a chronic inflammation, the coats weaken and bulge, causing a posterior staphyloma and a great increase in the myopia. Myopia of twelve dioptries and over is by no means uncommon, and in such cases no glasses will bring back clear distant vision, for pathological changes have already taken place in the eyeball. In children and young people myopia should be measured when the eye is under the influence of atropine, for it sometimes appears greater than it really is, on account of spasm of the ciliary muscle. The first and most important treatment of myopia is its correction by glasses. Much difference of opinion exists as to the strength of glasses which should be given for different degrees of myopia. This is not the place to enter on this discussion, so I shall only give the method I adopt myself, which

seems to me to recommend itself, both on physiological and practical grounds. In young people or children, if the myopia is not more than 4 or 5 D, glasses should be given which fully correct it, and should be worn constantly, at least till education is completed. In higher degrees the highest glasses should be ordered, with which the patient can see comfortably—objects not appearing much smaller than natural. These glasses too should be worn constantly. In adults myopia of 2 or 3 D may only require glasses for occasional use, so that “folders” of the strength most pleasant for the patient should be ordered. In higher degrees glasses may be worn constantly, and in myopia of say 8 or 10 D, two pairs of glasses are useful—one as high as can be worn with comfort for distant vision, and one about 3 or 4 D less for work. In the case of young myopes, where there is risk of the myopia increasing, care should be taken as to good light and upright position at work, the head not being bent down, and the general health attended to.

Astigmatism is an error of refraction caused by a departure from the normal spherical shape of the corneal surface, its curvature being greater in one direction than in another, so that no object can ever be accurately focussed on the retina, for if in focus in one meridian, it is out of focus in another, almost always at right angles to it. If the refraction is normal in one meridian and abnormal in another, it is called simple astigmatism—either hypermetropic or myopic, according to the refraction in the abnormal meridian. If both meridians are myopic, one more than the other, it is compound myopic astigmatism; and if both are hypermetropic, compound hypermetropic astigmatism. If one is hypermetropic and the other myopic, it is mixed astigmatism; and, lastly, if the two meridians differing to the greatest extent are not at right

angles, and refraction varies all over the cornea, it is irregular astigmatism.

*Symptoms* vary greatly according to the degree of astigmatism present. More or less asthenopia — pain and discomfort in seeing—is almost always found, and frontal headache is a very constant symptom. Sometimes, indeed, it is the only one. If the sight of each eye singly is tested with the astigmatic fan, sold by all opticians—a simple diagram consisting of rays radiating from a centre to the circumference of a semi-circle—it will be found that some rays appear blacker than others, the darkest and lightest corresponding with the two diameters of the cornea whose curvature differs to the greatest extent. In minor degrees the lens may neutralise the error by irregular curvature caused by irregular contraction of the ciliary muscle, but this tires the muscle greatly, and causes symptoms like those of hypermetropia, in an aggravated form. On examining the sight, distant vision is found to be more or less weak, and no ordinary spherical glasses can be found to bring it up to normal. In all such cases in young people an objective examination by the shadow test should be made under atropine. If astigmatism is present, different lenses will be needed to reverse the movement of the shadow in different meridians, and the two differing most widely, at right angles to each other, are measured. These are not necessarily the vertical and horizontal meridians, but may be any two at right angles to each other. In young people it is best to make an objective examination and confirm by a subjective with cylindrical lenses; but in adults the subjective alone is often sufficient. The subjective examination of astigmatism is a very tedious affair, for many combinations of spherical and cylindrical glasses must be tried to find which is best. The glasses finally ordered are chosen just as in hypermetropia or myopia,

with the addition of such cylindrical lenses as give best vision. These will generally be rather less than the strength found by the objective examination. The effect of the glasses must not be judged of too soon ; they may have to be worn for some weeks before the patient gets accustomed to them. Young people should wear them constantly, as taking them off and on only tires the ciliary muscles. Few general practitioners care to undertake the difficult task of the measurement and treatment of astigmatism, but it is, nevertheless, important that all should be sufficiently familiar with it to bear in mind the possibility of its presence as the cause in chronic headache, or even, according to some authorities, in epilepsy.

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## CHAPTER XI.

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### AFFECTIONS OF THE EYE IN RELATION TO OTHER DISEASES.

Though the connection between the affections of the eye and more general diseases is often mentioned under various headings in other chapters, it will be convenient and useful to have a brief summary of such connections, since in many cases they are of great importance, and yet often overlooked.

Acute Specific Fevers are often accompanied by Catarrhal Conjunctivitis, as is most frequently seen in Measles. Metastatic abscess may occur in the cornea in the desiccation stage of Smallpox, often in both eyes. Such abscesses break down into ulcers, and destroy vision. Happily they

seldom or never occur in cases where the patient has been partially protected by vaccination in infancy. Optic atrophy, either primary or consecutive, is sometimes seen after febrile diseases, more often after typhoid than any other. In rare cases neuritis may occur without atrophy. Embolism has been seen in Typhoid, and sometimes occurs in Pyaemia, when it is a most serious symptom (p. 56). Eye symptoms due to albuminuria or uraemia may occur after Scarlatina.

Syphilis presents many points of connection with eye disease. In acquired syphilis the primary sore has sometimes been found on the eyelids. Iritis is a most important manifestation of the secondary disease (p. 36). There is generally less pain and photophobia than in the rheumatic form, and it does not recur, but the diagnosis depends chiefly on the history. It occurs within a year of the primary sore, and both eyes are generally affected. Retinitis and choroiditis are sometimes seen about a year after the primary sore, but are rare. This form of choroiditis begins at the disc, and does not spread far from it. Rather later than this a form of choroiditis accompanied by gummata in the choroid is sometimes seen. In tertiary syphilis paralysis of special nerves occurs, causing different forms of ophthalmoplegia or paralysis of single muscles (Chapter XIII). Gummata are rare. Disseminated choroiditis (p. 59) with much atrophy is seen at a very late period. Keratitis is very rare in acquired syphilis. Inherited syphilis may cause disseminated choroiditis beginning at the periphery at a very early age—possibly *in utero*. Iritis is rare in this form of the disease, but when it does occur it often destroys sight by blocking the pupil with lymph. Most cases occur in infancy or early childhood, and more in girls than boys. Interstitial Keratitis is much the commonest manifestation of inherited disease in the eye (p. 30).

**Tuberculosis** is not a common cause of eye disease, unless one includes those strumous children suffering from Phlyctenular Ophthalmia (p. 22). Tubercles are occasionally, but rarely, seen in the iris, retina, or choroid. Optic neuritis in children is generally due to Tubercular Meningitis.

**Rheumatism** is not directly associated with any eye affection, but episcleritis (p. 33) and iritis (p. 34) occur more frequently in those of a rheumatic or gouty constitution. Cases of ocular paralysis are often attributed to the same cause.

**Bright's Disease** has two most important points in connection with the eye. Albuminuric retinitis (p. 53) occurs in about 30% of the cases of chronic Bright's disease, and is often the first symptom noticed. When a sudden failure of sight occurs in one or both eyes in a middle aged or elderly person, the possibility of this should always be borne in mind, and the optic disc searched for neuritis, and the urine examined for albumin. The second point of connection is the amaurosis sometimes caused by uraemia (p. 71).

**Cerebral Hæmorrhage** is sometimes preceded by retinal hæmorrhage (p. 55). Loss of sight in one eye is not an uncommon occurrence in cerebral hæmorrhage, and may assist in localising the seat of the lesion. As a general rule, no signs are seen in the fundus. Paralysis from cerebral hæmorrhage may affect several of the muscles of the eye, but seldom one only.

**Cerebral Abscess** causes no special eye symptoms, but is sometimes associated with Optic Neuritis—specially if increasing quickly.

**Cerebral Tumour** is a frequent cause of Optic Neuritis (p. 64), the latter being an important sign of tumour, as it is present in at least 80% of cases, according to the best

authorities. When a diagnosis of tumour has been made, the course of the Neuritis may throw some light on the prognosis. If it advances rapidly, the cause is probably doing the same, whereas if it subsides before reaching an advanced stage, the probability is that the growth of the tumour has ceased. It has lately been noticed that optic neuritis has disappeared after trephining of the skull, even though the tumour had not been removed: this affords a strong argument in favour of the theory of its causation by pressure.

Meningitis, when occurring at the base of the brain, is a common cause of Optic Neuritis (p. 64). It is most frequently seen as Tubercular Meningitis in children, and in some cases the neuritis may aid in forming a diagnosis. Traumatic and Syphilitic Meningitis are both occasionally accompanied by optic neuritis, but in Cerebro-spinal Meningitis it is rare.

Transverse Myelitis has in a few instances been seen accompanied by eye symptoms:—Optic Neuritis and Atrophy, or Hemianopsia (p. 71).

Locomotor Ataxy (Posterior Sclerosis of the Cord, Tabes Dorsalis) is very frequently associated with Optic Atrophy (p. 66). The proportion of cases of the former in which the latter occurs as a symptom is probably about 15%, while the proportion of cases of the latter in which the former can be recognised as cause is about 50%. Where present it is generally an early symptom, and may even occur years before any other symptoms of spinal disease are seen. Transient diplopia (Chapter XIII.) is sometimes an early symptom of Locomotor Ataxy, and another frequent symptom is the so-called "Argyll Robertson phenomenon." In this the pupil reacts to convergence and accommodation, but not to light. This may be seen in various spinal diseases, but is most frequent in Ataxy.

Disseminated Sclerosis is occasionally accompanied by more or less impairment of vision, without ophthalmoscopic signs. More rarely there is Optic Atrophy, similar to that in Posterior Sclerosis. Nystagmus is sometimes seen.

Epilepsy has in some cases an undoubted connection with errors of refraction, and is improved, or cured, when such errors are corrected by suitable glasses. The frequency of such cases is a hotly contended point, some—Americans specially—teaching that a very large proportion of cases of epilepsy are due to refractive defects, while British authorities are less positive on the point. It is, however, quite safe to say that they are sufficiently frequent to warrant us in making a careful examination of the refraction in every case of epilepsy coming under our notice, and correcting any error that may be present.

Migraine and Headache are not unfrequently associated with refractive defects, and an examination for such should be made in chronic cases which do not prove amenable to general treatment. Astigmatism (p. 82) is a specially common cause of headache, and every ophthalmic surgeon has seen cases of this which have come to him on account of defective vision, and on proper glasses being prescribed, the patient has been astonished to find that his long standing chronic headache, the cure of which he has quite despaired of, has entirely disappeared. Naturally, in such cases, the family physician comes in for some disrespect, which he would have avoided if it had only occurred to him to suggest defective sight as a possible cause of the headache.

Hysteria is a frequent cause of eye symptoms, specially transient Amblyopia, Amaurosis, Hemianopsia, &c.

Exophthalmic Goitre does not cause any change in the structure of the eye, but displaces the whole globe outwards. In some severe cases, the exophthalmos may be

so great that the lids can not meet over the eye, and the consequent exposure causes inflammation of the conjunctiva or cornea.

**Diphtheritic Paralysis** may affect the eye, causing paresis of the ciliary muscle, or it may affect one or more of the orbital muscles. For the general question of ocular paralysis see Chapter XIII.

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## CHAPTER XII.

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### INJURIES TO THE EYE.

**Burns** are not unfrequent among those whose work necessitates the handling of acids, quick lime, and other corrosive substances, and are also seen, though less frequently, as the result of the direct action of heat from splashes of molten metal, &c. When caused by the former the first thing to do is to wash out the conjunctival sac with plenty of warm water. After that, in either class, the treatment consists in frequent bathing with warm antiseptic lotions, and the use of drops, either of cocaine or opium, to relieve the pain. If the burn is extensive it must be carefully watched, to see that the wounded surfaces do not adhere, causing symblepharon—adherence of the lid to the globe.

**Foreign Bodies** becoming lodged under the lids, or on the cornea are a very frequent source of trouble. The history of the case generally points to the origin, but where such is not the case the appearances commonly

give a clue. A superficial inflammation of some part of the eye, which neither spreads nor disappears, should always suggest the possibility of a foreign body, and cause a careful search to be made. This is specially the case with an apparently obstinate catarrhal conjunctivitis in one eye only, in a child, which may very likely be due to a foreign body under the lid. When a foreign body is discovered its removal is simple, if on the lids. If on the cornea, care is necessary. The eye should be cocainized with a few drops of a 2% solution, or one of the several forms of tabellae. The surgeon stands behind the patient, and steadying the eye with a finger above and below, removes the foreign body with a small spud. In a few cases, where it has penetrated a little way into the cornea, a sharp flat needle may be required, but this must be carefully used, so as not to perforate the cornea. The patient should always be warned that pain and uneasiness will probably continue a day or so after the removal of a foreign body from the cornea. A drop of castor oil on the wound tends to allay pain.

**Blows on the Eye** may produce any effect, from temporary pain or sub-conjunctival ecchymosis to complete destruction of the globe. Two serious results are liable to follow a severe blow—dislocation of the lens, complete or partial; or rupture of a vessel, and intraocular hæmorrhage. The former may be hard to detect, unless the eye is seen immediately after the blow, for the pupil soon becomes dilated, and it is only by its tremulousness at one point that a partial dislocation can be detected. Very severe pain lasting for some hours after the blow should cause suspicion of dislocation. Intraocular hæmorrhage is plainly seen if it occurs from the iris into the anterior chamber. If from the fundus it causes more

or less complete loss of sight, and of the ordinary ophthalmoscopic reflex, and raising of the tension of the eye. Rest in a dark room should be ordered, and the course of events watched. Cataract may follow, if the capsule of the lens has been ruptured, or glaucoma, if there has been severe hæmorrhage into the globe. In still more severe blows, the globe may be ruptured, in which case enucleation is generally necessary.

**Penetrating Wounds of the Eye** are a source of great danger to the sight of the patient, and of great anxiety to the surgeon, as the possibility of sympathetic ophthalmia so often comes up. If the wound is a clean one, and only corneal, the danger is not great. When the iris has not prolapsed, eserine is used if the wound is peripheral, or atropine if it is central, to prevent prolapse. When prolapse has taken place, an attempt should be made to replace the iris, if seen immediately. If this attempt does not succeed, or if the case has not been seen early enough for it to be made, the prolapsed portion should be excised, and the edges freed from the wound. If the prolapse has existed several days when seen it is better to leave it alone, simply applying a firm pressure bandage to the eye, and if signs of dragging on the iris appear, doing an iridectomy. Wounds passing outside the cornea, and involving the ciliary region, are certainly more serious than purely corneal ones, though it is probable that the difference in severity has been rather exaggerated, and that this depends more on the cleanliness or otherwise of the wound than on its position. The great difficulty in these cases is to answer the question whether the risk of sympathetic ophthalmia is so great as to justify us in sacrificing the wounded eye, and the chance of retaining useful vision in it. As far as possible the question should

be fully and plainly stated to the patient or his friends, and the decision left to them ; but the medical man will generally be pressed for a very definite opinion, and even when an ophthalmic surgeon has been called in the family physician will be expected, and should be prepared, to give an opinion. No hard and fast rules can be laid down, for every case must be judged separately, but certain general principles should be kept in mind. If the wound is

1. very extensive, and a considerable part of the contents of the globe have escaped, no sight can be expected, and the eye should at once be enucleated or eviscerated (*vide infra*).
2. If a foreign body has lodged in the eye, an attempt should be made to remove it with an electro-magnet if it is iron or steel. If this fails we may wait a day, and if there is no sign of inflammation, and a fair amount of sight remains, a chance may be given, otherwise enucleation or evisceration is indicated. If, at any stage, pressure over the ciliary
3. region causes pain, and the eye is soft and shrinking, its removal is advisable. Lacerated and septic wounds are far more likely to be followed by sympathetic ophthalmia than clean cut and clean wounds, and the risk is also increased by loss of vitreous. Supposing it is decided to give the wounded eye a chance, it must be carefully watched, and if
4. any signs of septic inflammation are seen in it, it should be at once removed. These signs may be a tendency to the extension of the inflammation to other parts of the eye, a general turbidity of the contents of the anterior chamber, and the appearance of a purulent character in the effused lymph. If a general suppurative inflammation—panophthalmitis—starts in the wounded eye, the other is not likely to suffer, and it is wiser to delay removal till the acute inflammation has subsided, for cases are on record where it has extended to the meninges along the optic

nerve after enucleation. If the wounded eye is removed within 24 hours of the injury, the risk of sympathetic inflammation is nil, and if within a week it is exceedingly small. If removed after this sympathetic inflammation may still arise in the other eye, though no signs of it were visible when the wounded eye was removed. If, however, an interval of a month elapses after removal without signs of mischief in the other eye it may be considered practically safe, and after two months perfectly so. In all the above cases, the operation of evisceration of the globe may be substituted for its enucleation. This operation consists in cutting away the cornea, thoroughly scraping out all the contents of the globe, inserting a glass ball in the cavity, and stitching the sclerotic over it. The advantage of this procedure is that it gives a firm bed for an artificial eye, and as the muscles are left intact the natural movements of the globe are retained. Where sympathetic inflammation has already started in the other eye, enucleation is to be preferred, but in other cases this operation may be substituted.

**Sympathetic Ophthalmia** is the name given broadly to any deep seated inflammation in an eye, caused by previously existing inflammation in the other eye. The usual form which this takes is a chronic irido-cyclitis, leading to complete destruction of the eye.

*Symptoms*—The onset is generally slow, and often very insidious. There is generally a prodromal stage, which may last even for years, but is oftenest followed in a few days or weeks by actual inflammation. This stage is characterised by slight irritability of the eye ; it is very sensitive to light, easily waters, and when used for near work, vision every now and then becomes suddenly dim from failure of accommodation. This stage is described as

**Sympathetic Irritation**, and sometimes passes off without a serious sequel, specially if the other eye is removed at the earliest sign of mischief in the sound eye. If, as usually happens, the irritation passes into inflammation, pericorneal injection is seen, the aqueous becomes turbid, the vitreous filled with floating opacities, the iris muddy and rotten looking, and the pupil filled with lymph. If enucleation of the other eye is early performed, these symptoms may subside without materially injuring the eye, but if they run on unchecked, total loss of sight, with softening and atrophy of the globe eventually occur.

*Pathology*—It was long thought that nervous irritation accounted for this affection, but there is now little doubt that it is caused by the conveyance of the products of inflammation—chiefly micro-organisms—from the wounded to the sound eye, through the lymph channels of the optic nerves. This accounts for the fact that it is caused chiefly, if not entirely, by perforating wounds of the eye through which the organisms gain admission. It also accounts for the fact that enucleation does not always prevent it, for the infection may already be in the lymph channels. The fact that it does not follow suppurative inflammation is said to be due to the early blocking of the lymph channels in such inflammation.

*Treatment*—For the question of enucleation *vide supra*. If a case presents itself in which sympathetic inflammation has reached an advanced stage, so that sight in the sympathising eye is almost or entirely lost, while the wounded eye still retains some useful vision, it is better to remove the sympathising eye, and not the wounded one, which is likely to be the more useful of the two. When in an early stage, whether enucleation has been done or not, the sympathetic inflammation should be treated with

mercury internally, and atropine locally, as in ordinary irido-cyclitis. The benefit of mercurial treatment is often very marked indeed. It may be carried out by inunction, a piece of Ung. Hydrarg. about the size of a bean being rubbed into the thigh or arm each night.

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## CHAPTER XIII.

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### THE OCULAR MUSCLES.

This is a subject which presents considerable difficulties to the student, but these difficulties will be reduced to a minimum by a careful study of the normal mode of action of the different muscles. The normal position of the eyes at rest is with their axes parallel, and directed straight forward. When any muscle acts a movement takes place, and the optic axis is turned in some other direction, but all the muscles except the lateral recti also cause a rotation of the eye, so that what was the vertical diameter of the cornea becomes rotated away from the vertical. In describing this rotation we speak of the vertical line being rotated outwards or inwards, and *always understand that we refer to its upper extremity*. The action of the different muscles is as follows, and will be easily understood by referring to a diagram of the muscles in a text book of anatomy :—

The Rectus Externus moves the cornea outwards.

The Rectus Internus moves the cornea inwards.

The Superior Rectus moves the cornea upwards and inwards, and rotates the vertical axis inwards.

The Inferior Rectus moves the cornea downwards and inwards, and rotates the vertical axis outwards.

The Superior Oblique moves the cornea downwards and outwards, and rotates the vertical axis inwards.

The Inferior Oblique moves the cornea upwards and outwards, and rotates the vertical axis outwards.

In each case the first movement is the chief. When the eye is directed inwards or outwards the corresponding rectus alone is in play. When upwards or downwards the corresponding rectus and the opposite oblique are acting, and when in any position between, as upwards and outwards, for instance, all the muscles used in either of these directions singly are in play. When the eye is directed in any way except straight up or down, or to right or left, the vertical axis becomes more or less tilted, and its direction may be remembered by the simple rule that *the end of the vertical axis nearest to the normal position of the corneal centre always tends to incline towards that point*. For instance, if the eye is directed upwards and outwards, the vertical axis is rotated outwards; if the eye is directed downwards and outwards the vertical axis is rotated inwards.

**Paralysis of Ocular Muscles—Occurrence—**Paralysis may affect one or more muscles of the eye. If only one muscle is affected it is oftenest the superior oblique or external rectus, as each has a nerve for its own supply; the cause may then be central. If one of the other muscles is affected alone the cause is never central, but must be in the course of the third nerve after it has divided for the supply of the different muscles. When several muscles are affected they are generally some or all of those supplied by the third nerve (*vide infra*).

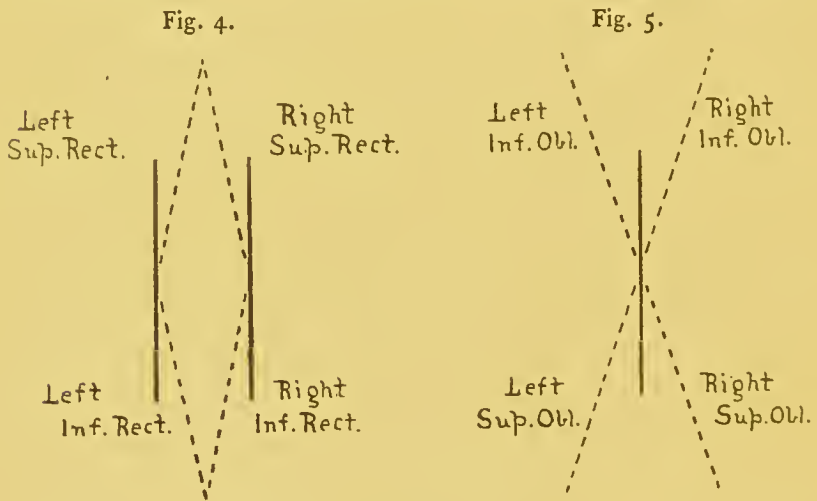
**Cause—**Ephemeral paralysis, first of one muscle then of

another, is sometimes seen in the preliminary stage of locomotor ataxy. In this case the lesion is central. The most common cause, however, is a central lesion due to syphilis. Almost any brain disease or injury may be accompanied by a lesion causing ocular paralysis. Diphtheria accounts for some cases. In others, which are probably of peripheral origin, the only cause appears to be cold, and these are classed as rheumatic. Any lesion in the orbit or about the base of the brain affecting a nerve in its course to the eyeball, may cause ocular paralysis.

*Symptoms*—Diplopia or double vision is the chief symptom, but in some cases of partial or slight paralysis the two images may be so near each other as to cause merely indistinct vision. Diplopia is usually accompanied by giddiness, so that the patient has to keep one eye covered. If the paralysis lasts some time the opposed muscle or muscles gradually contract, pulling the eye more and more away from the side of the paralysed muscle, and causing a perfectly evident strabismus, but in many cases the change in the direction of the eye is so slight as to escape notice at first. In these old cases diplopia often disappears, owing to supression of the image in the weaker eye.

*Diagnosis*—This depends chiefly on the relative positions of the two images, which we test by holding a lighted candle at a few yards from the patient, covering one eye with a coloured glass, and noting how he describes what he sees when the candle is held first in front, and then to right and left, above and below. If the image in the left eye is seen to the left, and the right to the right, it is homonymous diplopia, if *vice versa*, crossed diplopia. If one external rectus is paralysed the eye turns in, and

homonymous diplopia results, the two images being on the same level, for this muscle causes no rotation of the vertical axis (*vide supra*). If an internal rectus is paralysed the eye turns out, and crossed diplopia results, the two images being parallel, and on the same level. But in paralysis of any other muscles the images cease to be parallel, and are at different levels on account of the rotation of the vertical axis caused by those muscles. In all cases the two images are more widely separated when the patient looks in such a way as should bring the paralysed muscle into action, for naturally its loss is then most noticed. The relative positions of the two images have been shown by many schemes and diagrams, the best being that by



Dr. Werner, of Dublin. The image seen with the sound eye—the “true image,” as it is called—is shown by a plain line, and the image seen with the paralysed eye—the “false image”—is shown by the adjacent dotted line. For instance, in paralysis of the right inferior rectus, by looking at fig. 4 we see at once that the true image is—as

it always is—upright, and the false image is to the left, at a lower level and inclined towards the true. The diagram also shows in what direction the separation becomes greatest, for in the muscles, whose names are *above*, it is greatest on looking up, and in those whose names are below, it is greatest on looking down. Thus in our instance, the separation of the images would become greater when the patient looked down, for in this direction the inferior rectus should come into play, if not paralysed.

*Treatment.*—If the paralysis can be traced to any constitutional cause, treatment should be directed first against this. Galvanism and massage may be tried locally if the paralysis has lasted some time. Prismatic glasses are of no use for constant wear, for while their effect is constant, the amount of diplopia varies with the movements of the eyes. They may, however, give great relief and help to a patient for one particular action, reading especially. If they are not worn the paralysed eye must be covered by a patch or dark glass, to prevent annoyance from diplopia.

**Ophthalmoplegia.**—When the whole third nerve is paralysed, complete oculomotor paralysis, or *ophthalmoplegia totalis*, results. This is very easily recognised. The lid droops over the eyeball, which is a little further forward than usual, owing to the slackness of the muscles. The eye is directed outwards and downwards, by the unopposed action of the two active muscles—the external rectus and superior oblique—which are not supplied by this nerve. The pupil is dilated, and all power of accommodation lost. This form of paralysis may affect both eyes.

*Ophthalmoplegia externa* is commoner than the last. It presents the same appearance, except that the two internal muscles of the eye—the ciliary muscle and the sphincter of the iris—are unaffected. The reason of this is that the

nucleus of the third nerve is somewhat spread out on the floor of the Sylvian aqueduct, and the most anterior part of it is that controlling these internal ocular muscles—hence disease which affects the rest of the nerve may leave them untouched.

*Ophthalmoplegia interna* is the converse of the last, affecting the internal muscles only. It is rare from natural causes, but is produced artificially by atropine.

**Conjugate Paralysis** is loss of power, not of any special muscle or muscles, but of associated movement of the eyes in some special direction. It is not common, but is most often seen in the lateral movements. The eyes will then lose all power of turning to the left, for instance, but that the individual muscles are not both paralysed is proved by the fact that in this case the right eye can be turned to the left in convergence. The explanation of this form of paralysis (which, however, is not universally accepted) is that in conjugate movements of the eyes the internal rectus is not innervated from the nucleus of the third nerve, but that it receives special fibres from the nucleus of the sixth of the opposite side, through which it receives an impulse to contract simultaneously with the external rectus of the opposite side, to bring about a conjugate movement of the eyes to the side of the latter. So, when the external rectus for the left side, for instance, is paralysed, the internal rectus of the right side may lose its power of associated action with it, for the movement of both eyes to the left. The lesion causing this is of course central. Not uncommonly it affects some other nerve as well; oftenest the fifth, whose nucleus is close to that of the sixth in the floor of the fourth ventricle.

**Nystagmus** is the name given to a peculiar condition of the eyes characterised by short, sharp movements of both

eyes, quickly repeated in the same direction, generally from side to side. It is generally associated with amblyopia, either as cause or effect; in the latter case this amblyopia is congenital, or has arisen in early youth. This amblyopia may be due to a defect of refraction, or to some defect of development, such as albinism, or to opacity of the cornea from inflammation.

Where the nystagmus precedes the amblyopia, it is due to some peculiar strain of the eyes, and is most frequently seen in coal miners who work for hours in dim light with their vision directed upwards. These are the only cases in which treatment is of any avail. Complete removal of the cause is often followed by great improvement or cure.

Nystagmus is sometimes observed as a symptom of disseminated sclerosis or cerebellar disease, or very rarely of cerebral disease.

**Squint, or Strabismus.**—Squint consists in a deviation of one eye from its normal direction, and is distinguished from paralysis by the fact that this deviation persists, no matter in what direction the patient looks, whereas in paralysis the deviation increases as the eyes turn towards the side of the paralysed muscle, and may disappear entirely when they are turned towards the other side. Diplopia is not present except sometimes when a squint is just coming on. The reason of this is that the squinting eye is generally defective to begin with, and the image in it is "suppressed"—that is, simply ignored after a little time, just as one learns to ignore the image seen with one eye when working with the microscope with both eyes open, as one should always do.

Squint is seen in several forms. The commonest is convergent strabismus, in which one eye turns in. In divergent strabismus one eye turns out; in alternating

strabismus first one eye and then the other turns in, but never both together. Any of these forms may be constant or periodic; they generally begin as periodic, and if unchecked become constant.

*Causes.*—In all cases of squint there is probably a latent cause existing, in an unequal balance of strength in opposing muscles, and then some exciting cause brings on the squint. The commonest exciting cause is an error of refraction. Three-fourths of all cases of convergent squint are due to hypermetropia (often combined with astigmatism), and two-thirds of all cases of divergent squint are due to myopia. In these cases the squinting eye will generally be found to show a greater error than the other. With or without an error of refraction, defective vision will often be found in the squinting eye, from opacities in the cornea due to phlyctenular ophthalmia or other disease, or possibly there may be congenital amblyopia.

The explanation of the association of convergent squint with hypermetropia, and divergent with myopia, is simple. In the former the hypermetrope, to see near objects, has to make a vigorous effort of accommodation, and consequently of the associated movement of convergence, and the internal recti become abnormally strong and overbalance the external. In the latter, on the contrary, the myope has to make little or no effort of accommodation, and convergence is not properly exercised, so that the internal recti becomes weak, and are overbalanced by the external.

*Treatment* is first non-operative, and then, if necessary, operative. In all cases the refraction should be carefully examined under the influence of atropine, and glasses ordered to correct any error present. Weak atropine

drops should be used for the first few weeks that the glasses are worn, and the *good* eye bandaged for half-an-hour daily, in order to exercise the muscles of the squinting eye. This treatment is only successful as a rule in young patients, in whom the squint has only recently appeared. Operative treatment consists in dividing the offending rectus—the internal in convergent squint, the external in divergent. In bad cases of the former it may be necessary to divide both internal recti, and even to advance the external. In divergent squint it is generally necessary to advance the internal rectus as well as divide the external, but treatment in these cases is seldom satisfactory. Operative treatment should not be carried out in children under 8 or 10 years of age, but non-operative treatment patiently persevered in. It only very rarely happens that a child “grows out of” a squint without proper treatment.





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